NEUROMUSCULAR HAMSTRING FUNCTION IN RESPONSE TO INTERMITTENT RUNNING AND PREVIOUS INJURY.

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ABSTRACT

Hamstring strain injuries (HSIs) are the predominant injury sustained in a number of sports and are notorious for exhibiting high rates of reinjury. Despite the identification of the hamstrings as a muscle group prone to strain injury, for several decades the incidence and recurrence rates of HSIs have continued to remain high. This suggests that, currently, a number of factors implicated in the aetiology of HSIs have yet to be considered thoroughly.

Fatigue is often suggested as a causative factor given the increased prevalence of HSIs in the later portions of halves in soccer and rugby union matches. Whilst fatigue has long been known to reduce neuromuscular function, the specific impact of intermittent running on hamstring function has only recently begun to receive attention. Furthermore, a history of previous injury is consistently identified as the primary risk factor for future HSI. Although previous injury is considered to be an unalterable risk factor such a definition neglects the potential for maladaptation associated with a prior insult to negatively alter hamstring function and, by extension, increase the risk of future injury. The identification of maladaptations in response to injury is of great importance as it informs the rehabilitation specialist on aspects of hamstring function that require attention during convalescence.

Intermittent running has been found to result in preferential declines in eccentric hamstring strength, with little to no impact on concentric strength. As eccentric weakness is a known risk factor for HSI, the development of targeted interventions to reduce the loss of eccentric strength following intermittent running would be

expected to minimise the increased risk of sustaining a HSI with prolonged activity. However, before an intervention can be determined the mechanisms responsible for this contraction mode specific decline in function need to be determined. Similar to the impact of intermittent running on hamstring function, previous work has shown that a history of HSI results in chronically lower levels of eccentric hamstring strength, despite athletes being sufficiently rehabilitated to return to training and competition. Once again, the impact on concentric hamstring function is minimal. The purpose of this doctoral thesis was to firstly examine the potential mechanisms responsible for the eccentric specific alteration in hamstring function following intermittent running (Study 1) and previous injury (Study 2 & 3), respectively.

The focus for study 1 was to determine if muscle damage was partially responsible for the loss of hamstring function following intermittent running. To test this, knee flexor angle of peak torque was used as a marker for the propensity of muscle to be damaged, as per the 'sarcomere popping' hypothesis. Marked variability was displayed in the cohort with regards to the decline in eccentric knee flexor torque lost following the running protocol with both low and high responders identified. There was, however, no difference in the knee flexor angle of peak torques between the two groups.

In studies 2 and 3 the examination centred on the impact of a prior HSI on the neuromuscular function of the knee flexors. Using a retrospective study design, athletes with and without a history of unilateral HSI had their maximal concentric and eccentric knee flexor strength and hamstring activity assessed using isokinetic

dynamometry and surface electromyography (sEMG) respectively. All athletes recruited for the injured group had a history of biceps femoris long head (BF_L) strain injury. The findings reported from study 2 showed that recreational level athletes who had previously sustained a HSI displayed knee flexor weakness during concentric and eccentric contraction modes compared to the contralateral uninjured limb. However, the sEMG activity data revealed that only during eccentric contractions did the previously injured BF_L display significant reduction compared to the uninjured limb. In study 3, a similar cohort was assessed, to determine whether more functionally relevant markers of neuromuscular hamstring function differed in previously injured compared to uninjured hamstrings. It was found that during eccentric contractions at $-60^{o}.s^{-1}$, knee flexor rate of torque development (RTD) and impulse (IMP) were significantly lower in a limb that had sustained a HSI compared to the contralateral uninjured limb 50 and 100ms after the onset of torque generation. Furthermore the associated early onset sEMG activity of the previously injured BF_L was lower in a limb that had sustained a HSI compared to the contralateral uninjured limb 100ms after the onset of sEMG activity during contractions at $-60^{o}.s^{-1}$ and $180^{o}.s^{-1}$.

The observations from the first three studies provided the impetuous for the final experimental study of this thesis. At present the gold standard measure for eccentric knee flexor strength, an isokinetic dynamometer, is largely inaccessible due to the high purchase price (>USD$70,000) and expertise required to run the device. These limitations severely reduce the number of athletes that have their eccentric hamstring strength assessed. A cheaper, field based alternative to isokinetic dynamometry

would be desirable as it would allow for more athletes to be assessed for eccentric strength deficiencies and would potentially allow for serial testing throughout a competitive season and/or pre-season. An experimental field testing device for the assessment of eccentric hamstring strength, utilised the Nordic hamstring exercise (NHE), was devised to circumvent the inaccessibility of isokinetic dynamometry. The purpose of study 4 was to determine the test-retest reliability of this device. Following the reliability aspect of study 4, a retrospective study was performed whereby a sub group of elite athletes with a history of HSI within the last 12 months had their eccentric knee flexor strength and between limb strength asymmetry determined using the experimental device. The experimental device displayed high to moderate levels of reliability and from the retrospective part of the study the previously injured limbs were 15% weaker than contralateral uninjured limbs, which is similar to previous observations (see Study 2).

This thesis has contributed to the current evidence base on the neuromuscular function of the hamstrings following intermittent running induced fatigue and prior HSI. Further to this, a potential solution to the eccentric specific nature of the current problem, in the form of a more accessible device to measure eccentric knee flexor strength, is presented. These findings inform that current rehabilitation practices for HSIs are inadequate at returning a previously injured limb back to optimum function and seems to neglect the role of the nervous system during convalescence. Further, the identification of high variability in eccentric knee flexor strength following intermittent running is suggestive that a high tolerance to fatigue might be beneficial for HSI avoidance. Practically this thesis highlights the need for greater attention to

be paid towards eccentric knee flexor and hamstring function in response to fatigue and previous injury. This would be expected to assist with a reduction in HSI and reinjury rates over time.

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ABBREVIATION

ACL	anterior cruciate ligament
BF	biceps femoris
BF_L	biceps femoris long head
BF_S	biceps femoris short head
EMG	electromyography
ES	Cohen's d effect size
H:Q	hamstring to quadriceps strength ratio
$H:Q_{conv}$	conventional hamstring to quadriceps strength ratio
$H:Q_{func}$	functional hamstring to quadriceps strength ratio
HSI	hamstring strain injury
ICC	intraclass correlation coefficient
IMP	impulse
MH	medial hamstrings
NHE	Nordic hamstring exercise
$^{o}.s^{-1}$	degrees per second
RTD	rate of torque development
sEMG	surface electromyography
SM	semimembranosus
ST	semitendinosus
TE	typical error
%TE	typical error as a co-efficient of variation
95%CI	95 percent confidence interval

STATEMENT OF ORIGINAL AUTHORSHIP

The work contained in this thesis has not been previously submitted to meet requirements for an award at this or any other higher education institution. To the best of my knowledge and belief, the thesis contains no material previously published or written by another person except where due reference is made.

Signature: 

Date: 11/13

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Published papers.

Opar DA, Williams MD, Shield AJ. Hamstring strain injuries: factors that lead to injury and re-injury. Sports Medicine. 2012;42(3):209-226

Opar DA, Williams MD, Timmins RG, Dear NM, Shield AJ. Knee flexor strength and biceps femoris electromyographical activity is lower in previously strained hamstrings. Journal of Electromyography and Kinesiology. doi: 10.1016/j.jelekin.2012.11.004.

Opar DA, Williams MD, Timmins RG, Dear NM, Shield AJ. Rate of torque and EMG development during anticipated eccentric contraction is lower in previously strained hamstrings. American Journal of Sports Medicine. 2013; 41(1):116-25.

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Opar DA, Dear NM, Timmins RG, Williams MD, Shield AJ. *Previous hamstring strain injury reduces knee flexor strength and biceps femoris activation.* Presented at the 59th Annual Meeting and 3rd World Congress on Exercise is Medicine of the American College of Sports Medicine, San Francisco, United Sates. 2012.

Opar DA, Dear NM, Timmins RG, Williams MD, Shield AJ. *Knee flexor strength and biceps femoris activation deficits following hamstring strain injury.* Presented at the XIX Biennial Conference of the International Society of Electrophysiology and Kinesiology, Brisbane, Australia. 2012.

Opar DA, Williams MD, Timmins RG, Dear NM, Shield AJ. *Impact of previous hamstring strain injury on rate of torque and EMG development during eccentric contraction.* Australian Conference of Science and Medicine in Sport, Sydney, Australia. 2012.

Patent applications.

Opar DA, Shield AJ. Apparatus and method for knee flexor assessment. International Patent Application No. PCT/AU2012/001041. 2012

1 GENERAL INTRODUCTION

Hamstring strain injuries (HSIs) are the most prevalent non-contact injury in Australian football,(1-7) American football,(8) rugby union,(9-12) soccer,(13-17) and sprinting.(18, 19) HSIs are characterised by acute pain in the posterior thigh with disruption of the hamstring muscle fibres(20) and range in severity from minor microscopic tearing and some loss of function (grade one) through to a full rupture of the muscle with complete loss of function (grade three).(21) The biceps femoris (BF) is the most commonly injured of the hamstring muscles(22-24) with the muscle-tendon junction and adjacent muscle fibres being the most common sites of disruption.(22, 25)

In many cases, HSIs cause considerable time lost from training and competition(7, 9, 15, 26) which results in financial loss(27) and diminished athletic performance.(28) Injury has been suggested to have cost in excess of £74.7m in English premier and football league clubs during the 1999-2000 season.(27) Similar estimates, made for this thesis, showed that for elite Australian football teams HSIs cost approximately $1.5m (Australian dollars) in the 2009 season, which represents 1.2% of the salary cap in the Australian Football League. Furthermore, player performance has been found to be significantly reduced following return from HSI in elite Australian footballers.(28)

Epidemiological data obtained from Australian football, rugby union and soccer across a number of years indicates that rates of HSIs have not declined in recent decades (Figure 1-1).(1, 2, 9, 13, 15, 17, 29) This is particularly worrying when

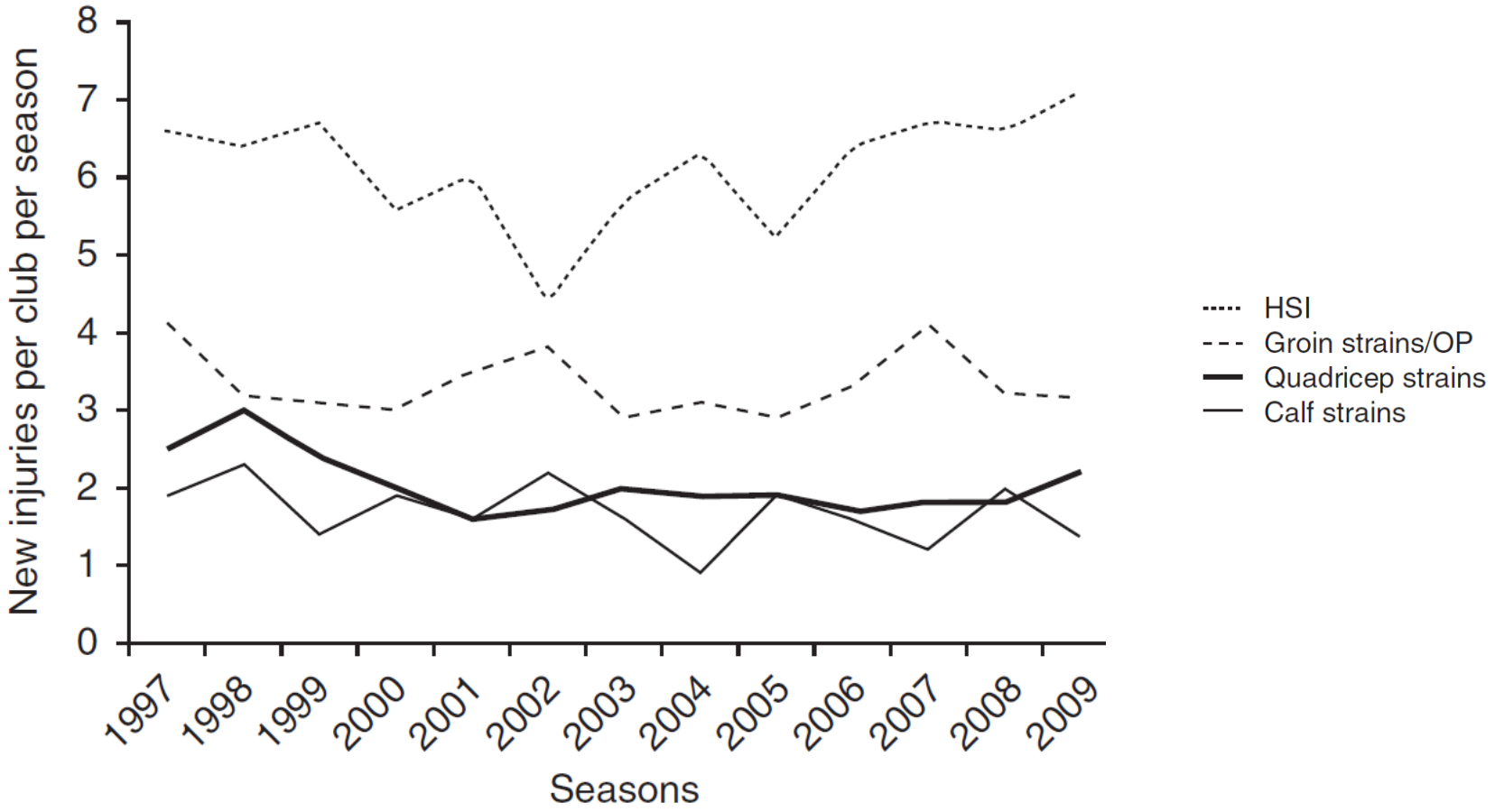

Figure 1-1. Injury incidence in the Australian Football League over 13 years. An injury is defined as 'any physical or medical condition that prevents a player from participating in a regular season (home and away) match' (adapted from Orchard & Seward,(7) with permission). HSI, hamstring strain injury; OP, osteitis pubis.

taking into account that HSIs have for a long time been a well documented problem which has received considerable attention in the literature. Moreover, other injuries, such as ankle sprains in soccer(30) and posterior cruciate ligament injuries in Australian football,(31) have shown reduced incidence rates following the implementation of relatively effective preventative measures. The lack of decline in HSI rates highlights that current practices aimed at preventing them requires further scientific investigation. In particular, whilst a number of risk factors for HSIs have been identified, most notably lower levels of eccentric hamstring strength,(32, 33) the impact of intermittent running on neuromuscular function of the hamstrings and the potential role of the nervous system in strain injury aetiology has been largely overlooked.

The importance of these two potentially contributing factors should not be overlooked. HSIs are thought to most often occur during the terminal swing phase of high speed running(34-38) and have been shown to occur at a greater incidence during the later portions of a half of soccer(15) and rugby union.(9) This evidence combined suggests that the likelihood of HSI is greater during high speed running particularly when in a fatigued state. Indeed recent work has identified specific reductions in eccentric hamstring strength following intermittent sprint running(39, 40) which conforms with the literature indicating lesser eccentric hamstring strength is a risk factor for HSI.(32, 33) However, as yet, the mechanisms responsible for this eccentric specific decline in hamstring strength following repeat sprint running is yet to be determined. Such mechanistic investigations are important to assist in guiding future efforts aiming to develop effective interventions to minimise the impact of

intermittent running on eccentric hamstring strength. Similarly, previous HSI, which is consistently identified as the primary risk factor for future HSI,(6, 20, 41, 42) also appears to have a long lasting impact on hamstring function, particularly eccentric hamstring strength.(43-45) Importantly these deficits in eccentric hamstring strength are apparent even after athletes have been given clearance to return to pre-injured levels of training and competition.(43-45) At least one paper suggests that these post-HSI deficits in hamstring function are partly due to reductions in muscle volume(46), however this does not explain why strength is only reduced during eccentric contraction. Little attention has been paid to the potential for deficits in neural function following HSI as being responsible for these long-term declines in eccentric hamstring strength. Determination of any neural contribution to deficits in hamstring function following 'recovery' from a HSI is vitally important as it will provide information as to how current rehabilitation strategies might be changed to attend to this altered function.

Given the gaps in the literature relating to hamstring function following repeat sprint running and previous HSI, the primary aim of this thesis was to determine the impact of intermittent running and previous HSI on the neuromuscular function of the hamstring muscle group and the mechanisms responsible for these changes. A thorough review of the current literature can be found in Chapter 2, followed by experimental studies which directly address the primary aims of this thesis in Chapters 3-5. Observations from these initial experimental studies then generated secondary aim for this thesis, exploring the possibility of developing a field testing

device to assess eccentric hamstring strength (Chapter 6). Findings from all studies are then collectively discussed in Chapter 7.

2 LITERATURE REVIEW

Factors that lead to hamstring strain injury and re-injury.

This chapter is comprised of the following paper published in Sports Medicine:

Opar DA, Williams MD, Shield AJ. Hamstring strain injuries: factors that lead to injury and re-injury. Sports Medicine. 2012;42(3):209-226.

The authors listed have certified that:

1. they meet the criteria for authorship in that they have participated in the conception, execution, or interpretation, of at least that part of the publication in their field of expertise;
2. they take public responsibility for their part of the publication, except for the responsible author who accepts overall responsibility for the publication;
3. there are no other authors of the publication according to these criteria;
4. potential conflicts of interest have been disclosed to (a) granting bodies, (b) the editor or publisher of journals or other publications, and (c) the head of the responsible academic unit, and
5. they agree to the use of the publication in the student's thesis and its publication on the QUT ePrints database consistent with any limitations set by publisher requirements.

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2.1 OVERVIEW

Hamstring strain injuries (HSIs) are common in a number of sports and incidence rates have not declined in recent times. Additionally, the high rate of recurrent injuries suggests our current understanding of HSI and re-injury risk is incomplete. Whilst the multifactoral nature of HSI is agreed upon by many, often individual risk factors and/or causes of injury are examined in isolation. This review aims to bring together the causes, risk factors and interventions associated with HSI to better understand why HSI are so prevalent. Running is often identified as the primary activity type for HSI and given the high eccentric forces and moderate muscle strain placed on the hamstrings during running these factors are considered to be part of the aetiology of HSI. However the exact causes of HSI remain unknown and whilst eccentric contraction and muscle strain purportedly play a role, accumulated muscle damage and/or a single injurious event may also contribute. Potentially all of these factors interact to varying degrees depending on the injurious activity type (i.e. running, kicking). Furthermore, anatomical factors such as the biarticular organisation, the dual innervations of biceps femoris (BF), fibre type distribution, muscle architecture and the degree of anterior pelvic tilt have all been implicated. Each of these variables impact upon HSI risk via a number of different mechanisms, including increasing hamstring muscle strain and altering the susceptibility of the hamstrings to muscle damage. Reported risk factors for HSI include age, previous injury, ethnicity, strength imbalances, flexibility and fatigue. Of these little is known, definitively, about why previous injury increases the risk of future HSI. Nevertheless, interventions put in place to reduce HSI incidence by addressing modifiable risk factors have focussed primarily on increasing eccentric strength,

correcting strength imbalances and improving flexibility. The response to these intervention programs has been mixed with varied levels of success reported. A conceptual framework is presented suggesting that neuromuscular inhibition following HSI may impede the rehabilitation process and subsequently lead to maladaptation of hamstring muscle structure and function including preferentially eccentric weakness, atrophy of the previously injured muscles and alterations in the angle of peak knee flexor torque. This remains an area for future research and practitioners need to remain aware of the multifactoral nature of HSI if injury rates are to decline.

2.2 LITERATURE SEARCH

The articles selected for review were obtained via searches of MEDLINE and SPORTDiscus between 1966 and April 2011. The following keywords were searched in combination: ‘hamstring’, ‘knee flexor’, ‘muscle strain’, ‘injury’, ‘mechanism’, ‘risk factors’ and ‘prevention’. From the abstracts returned, articles were included for review if they related to hamstring injury incidence, causation, risk factor analysis or prevention. Full text copies of selected articles were then sourced and the reference lists of these articles were hand searched to identify other potential articles.

2.3 HAMSTRING STRAIN INJURY INCIDENCE AND RECURRENCE RATES

In track and field one group has reported that HSI account for 26.0% of all injuries sustained, with most occurring in sprinting events.(19) In comparison, observations from Australian football and soccer indicate that HSI are responsible for 13-15%(1, 2, 4) and 12-14%(13, 14, 16) of all injuries respectively. These figures are comparable to reports from American football training camps (12%)(8) and rugby union training (15%).(12) HSI are also the single largest cause of lost playing time in Australian football(7) and are the predominant injury type responsible for prolonged absence (> 28 days) from training and playing in soccer.(13)

When compared to earlier epidemiology data from Australian football,(2) rugby union(2) and soccer,(17) recent observations indicate that the incidence of HSI in sport has trended upwards over the past two decades. Further, data from the Australian Football League Annual Injury Report displays an increasing trend in the incidence of HSI over the past seven competitive seasons whilst other major injuries, including other prevalent lower limb muscle strains, have remained largely stagnant (Figure 1).(7)

In addition to high incidence rates and significant time lost, HSI also exhibits a very high rate of recurrence.(2, 7, 9, 15, 47-50) Over 13 seasons of observation, 27% of all HSI in the Australian Football League are recurrences of previous injuries, however recent evidence suggests this is trending downwards, arguably because of a more conservative approach in return to play strategies rather than improved

rehabilitation practices.(7) Similarly high rates of HSI recurrence have also been reported in American football (32%),(47) rugby union (21%)(9) and soccer (16%).(51)

2.4 HAMSTRING FUNCTION DURING RUNNING AND POTENTIAL FOR STRAIN INJURY

Although kicking, tackling, cutting and slow-speed stretching can result in HSI,(9, 15, 17, 24, 52) running accounts for the majority of HSI in soccer(15) and rugby union,(9) which suggests the demands of running give the greatest insight into the causes of HSI.

Studies of running biomechanics have found the hamstrings are active for the entire gait cycle with peaks in activation during the terminal swing and early stance phases.(38, 53) During the terminal swing phase the hamstrings are required to contract forcefully whilst lengthening to decelerate the extending knee and flexing hip.(34, 36-38, 54) It is also in terminal swing that the hamstrings reach their maximum length.(37, 38) Of the three biarticular hamstring muscles, biceps femoris long head (BF_L) undergoes the greatest stretch, reaching almost 110% of the length in upright standing during terminal swing whilst semimembranosus (SM) and semitendinosus (ST) reach 107.5% and 108.2% respectively.(37) In contrast, the maximum torques for hip extension and knee flexion are found to occur during ground contact in overground sprinting.(55) During this phase the hamstrings are acting primarily concentrically to extend the hip,(54) however it has been reported that an eccentric contraction of the hamstrings occurs during the late stance phase of overground sprinting.(38)

The presence of a high force eccentric contraction during the stance(38) and swing(34-38) phases likely contributes to the high rates of HSI during maximal

speed running. The terminal swing phase is considered the most hazardous as the hamstring muscle-tendon units are at their longest length of the gait cycle and are most heavily activated.(34-38) This suspicion has been supported by two independent serendipitous observations of acute HSI during biomechanical studies of running, the timing of which was consistent with the insult occurring in terminal swing.(35, 56) Whilst the stance phase is another possible period of susceptibility to HSI, due to high hip extension and knee flexion torque,(55, 57) it involves much shorter hamstring lengths compared to terminal swing.(34, 36-38)

2.5 CAUSES OF HAMSTRING STRAIN INJURIES

In addition to strain injuries the hamstrings are also affected by tendinopathies(9) and back related injuries that referred pain to the posterior thigh.(20) These injuries display varying aetiological characteristics and as such the causes of these injuries vary considerably. For the purposes of this chapter the focus will be on the cause of HSI during running.

There is some debate as to whether muscle strain or the magnitude of eccentric force is the causative factor in muscle strain injuries. Observations from *in-situ* animal models suggest that the magnitude of muscle strain is the primary determining factor in the occurrence of strain injury.(58-60) Many investigators have also suggested that *in-vivo* muscle strain injuries are associated with high force eccentric contractions,(34, 36, 38, 45, 60-66) where the lengthening demands placed on the muscle exceed the mechanical limits of the tissue.(34) It remains to be seen if both high eccentric force and high muscle strain are necessary conditions for a strain injury or whether each on their own is sufficient to bring about strain injury. Biomechanical observations suggest that eccentric contraction is a necessary condition for a HSI during running(35, 56) and this claim is strengthened by the lack of strain injuries in concentrically-biased sports such as swimming and cycling.(67, 68) An argument for muscle strain being a necessary condition is less clear given that HSI have been reported for both high (i.e. kicking)(9, 15) and low (i.e. sprinting)(9, 15, 19, 25) strain tasks. Potentially an interrelationship exists between eccentric force and muscle strain that dictates whether a muscle is injured. For example, strain injury

may be avoided in tasks that involve high levels of strain if the level of eccentric force is low and the same may be true for high eccentric force/low strain activities.

There is also some uncertainty as to whether HSI most typically occur as a result of accumulated microscopic muscle damage,(69) or as a result of a single event that exceeds the mechanical limits of the muscle.(70) It seems feasible, however, that both may contribute. For example, the accumulation of microscopic damage may leave the muscle tissue in a vulnerable state and more susceptible to injury in the event of a single traumatic event such as bending to pick up or catch a ball.

Whilst the potential role of accumulated muscle damage in muscle strain injury aetiology is not disputed, debate continues as to the physiological process responsible for damage. Morgan(69) first proposed the accumulated damage theory when he postulated that microscopic damage caused to individual sarcomeres following eccentric exercise was as a result of preferential lengthening of weaker sarcomeres. This theory suggests that during eccentric contractions there is non-uniform lengthening of adjacent sarcomeres when muscles are operating on the descending limb of the length-tension curve.(69) This difference in sarcomere length impacts upon force creating capabilities of sarcomeres as per the properties of the length-tension curve, which indicates that sarcomeres extended past their optimum length display a reduction in force generating capacity.(71) This results in weaker sarcomeres (i.e. sarcomeres longer than optimal length) lengthening uncontrollably during eccentric contractions and eventually being excessively stretched so that passive structures take up most of the tension due to the reduction in actin-myosin

overlap.(69) The consequential damage to individual sarcomeres as a result of this uncontrolled lengthening was termed ‘sarcomere popping’ and was proposed to be the first step towards macroscopic muscle damage such as muscle strain injury.(69)

Morgan’s hypothesis(69) is not, however, universally accepted. It has been criticised because it is based upon single myofibril stretch studies performed *in-vitro* and *in-situ* which involve fibre strains not considered to be within the physiological range.(72) Butterfield(72) also argues that the expectation of unstable sarcomere lengthening on the descending limb of the length-tension curve is flawed given that the length-tension curve is determined under isometric conditions, whilst muscle lengthening occurs during dynamic eccentric contraction. Indeed, evidence exists of inherent stability of the length-tension curve during lengthening contraction(60, 73) which is thought to be attributable to the physiological characteristics of titin.(72) Further evidence(74) also argues against the assertion that sarcomeres at a longer length will lengthen uncontrollably when exposed to eccentric contraction. This still remains an area of great controversy.

Our current understanding of HSI suggests that high levels of eccentric force(34, 36, 38, 45, 56, 57, 60-66) and muscle strain(58-60) are implicated in the aetiology of strain injury, however it is not clear whether accumulated microscopic muscle damage(69) or the presence of a single injurious event(70) are most typically responsible for injury. Potentially any one of these factors may be the primary cause of HSI depending on the injurious activity type. For example, muscle strain may be

the predominant mechanism in kicking HSI whereas forceful eccentric contractions may be the major mechanism in running HSI.

2.6 ANATOMICAL FACTORS THAT PREDISPOSE HAMSTRINGS TO STRAIN INJURY

The predominately biarticular nature of the hamstrings allows for simultaneous extension at the hip and flexion at the knee during concentric contraction and significant lengthening during concurrent hip flexion and knee extension as seen in running(54) and kicking.(75) Such lengthening demands are thought to predispose the hamstrings to strain injury as the lengthening may exceed the mechanical limits of the muscle(34) or lead to the accumulation of microscopic muscle damage.(69, 76)

The two heads of the BF muscle are innervated by different nerve branches; BF_L by the tibial portion of the sciatic nerve and the BF short head (BF_S) by the common peroneal branch of the sciatic nerve, and it has been suggested that this dual innervation is a possible explanation for HSI because of the potential for uncoordinated contraction of the two heads of BF.(15, 47) This, however, remains unsubstantiated and is yet to be the focus of scientific investigation. Another commonly held belief is that the hamstring muscles possess a high number of type II fibres(77) and this would be expected to increase the risk of strain injury given that fast glycolytic fibres have shown a greater propensity for muscle damage following eccentric contraction in animal models.(78) However, whilst early histochemical analysis suggested that the hamstrings consisted predominately of type II muscle fibres (58%)(77) a more recent study reported that only 51% of fibres in the BF_L were classified as fast twitch.(79) This discrepancy may be due in some part to the difference in the ages of the study participants. Subjects from the study by Garrett

and colleagues(77) ranged in age from 37-76 years whereas the hamstrings utilised in the study by Dahmane *et al.*(79) better reflected the ages seen in elite sport (17-40 years). Whilst fibre type distribution may be one factor that impacts upon the strain injury risk of muscles, it's role in HSI may have been overstated previously given the fact that the vastus lateralis has been shown to have a greater proportion of type II muscle fibres(80) compared to BF_L,(79) yet the hamstrings are more commonly injured than the quadriceps.(7, 11-13, 42) In this example the differing lengthening demands of the muscles may have a greater influence over the propensity for strain injury than fibre type distribution.

Variations in muscle architecture may also explain high rates of muscle-specific HSI. For example, BF_S possesses much longer fascicles but a much smaller physiological cross sectional area compared to BF_L(81) and this variation of architecture may predispose the BF, particularly the long head, to high rates of strain injury. Longer fascicles allow for greater muscle extensibility(72) and reduce the risk of over lengthening during eccentric contraction.(76) However BF_L, which undergoes the greatest lengthening of all the hamstrings during sprinting,(37) has shorter fascicles compared to BF_S and this may predispose the BF_L to repetitive over lengthening and accumulated muscle damage.(69, 76) Consideration must be given to the fact that the available hamstring architecture data from this cadaveric study(81) has been performed on muscles from donors aged 68-88 years and the architectural characteristics of these muscles may differ markedly from younger, athletic populations.

The degree of anterior pelvic tilt may also impact upon HSI risk given that the common origin for the long hamstrings, the ischial tuberosity,(82) is found on the posterior aspect of the pelvis. As a result, excessive anterior pelvic tilt will place the hamstring muscle group at longer lengths(83) and some have proposed that this may increase the risk of strain injury.(15, 84)

Whilst some commonly held beliefs relating to HSI risk, such as the importance of fibre type distribution, may now be questioned, the importance of structure still remains crucial to hamstring muscle function. As such, the anatomy of the hamstrings most likely contributes to its high propensity to injury, however each of the aforementioned anatomical variables may increase the risk of injury via discrete mechanisms. An understanding of each of these anatomical factors must also be intepreted with an understanding of the causes of HSI presented in Chapter 2.5.

2.7 RISK FACTORS FOR HAMSTRING STRAIN INJURIES

A number of unalterable and alterable risk factors have been proposed for HSI, including, but not limited to, increasing age,(6, 15, 16, 20, 41, 42, 85, 86) previous injury,(6, 20, 41, 42, 86) ethnicity,(9, 15, 20) strength imbalances,(5, 32, 33, 43, 47, 87-91) extremes of flexibility(44, 84, 92-95) and fatigue.(47, 59, 96, 97) This chapter details those prospective studies which have identified unalterable and alterable factors that elevate the risk of an athlete sustaining a HSI. In addition, both intervention studies and randomised controlled trials aimed at preventing HSI are examined to provide a thorough understanding of the alterable causative factors responsible for HSI.

2.7.1 UNALTERABLE RISK FACTORS

2.7.1.1 Age

Increasing age has been identified by a number of investigators as an independent risk factor for HSI in Australian footballers(6, 20, 41, 85) and soccer players.(15, 16, 42, 86) Australian footballers older than 23(41) or 24 years(6) and soccer players older than 23 years(15) are at an elevated risk of HSI, with the odds ratios as high as 4.4 (95%CI: 1.6 to 12.5) for the older athlete.(6) Furthermore, each year of age has been reported to increase the risk of sustaining a HSI by as much as 1.3 (95%CI: 1.1 to 1.5) fold in Australian footballers(20) and by 1.8 (95%CI: 1.2 to 2.7) fold in soccer players.(16) Importantly, all studies which report age as a significant risk factor have utilised regression or multivariate analysis to conclude that increasing age increases the risk of sustaining a HSI independently of confounding variables such as previous injury.(6, 15, 16, 20, 41, 42, 85, 86)

One attempt to identify age related changes that lead to an increased risk of HSI in Australian football identified increased body weight and reduced hip flexor flexibility as predictors of HSI in athletes aged 25 years or older.(85) Despite achieving significance, the increase in risk was moderate with risk ratios of 1.07 (95%CI: 1.0 to 1.2) and 1.15 (95%CI: 1.0 to 1.3) respectively.(85) Other suggestions are that decreases in muscle mass and strength due to ageing could partially explain the increased risk of HSI in the older athlete,(6) however evidence to support this hypothesis(98, 99) comes from cross sectional studies that included non-elite, non-athletic cohorts of significantly greater age ranges than are observed in elite sport. It is, in our view, particularly unlikely that athletes aged 24-30 are weaker or have less muscle mass than their 18-23 year old counterparts. Other hypotheses are age-related changes to muscle structure(6) and entrapment of L5/S1 nerve root due to hypertrophy of the lumbosacral ligament,(100) however more evidence is required to test these hypotheses.

Despite the consistent identification of age as a risk for HSI, no convincing explanation has been given as to why athletes older than 24 years are at significantly greater risk than younger athletes. Ideally long term longitudinal studies are required to determine the physiological changes that occur across an athlete's career to further elucidate the relationship between increasing age and increased HSI risk.

2.7.1.2 Previous injury

A number of studies have indicated that Australian footballers with previous HSI are at an elevated risk of sustaining a future HSI.(6, 20, 41) HSI from the previous season was also a significant risk factor for hamstring injury in elite professional soccer players(86) and has been reported to increase the risk of future injury as much as 11.6 (95%CI: 3.5 to 39.0) fold.(42)

Following a HSI the primary goal must be to identify the predisposing factor responsible for the injury, which then should be a target for rehabilitation and/or intervention.(49) If this predisposing factor is not ameliorated the athlete will remain at an elevated risk of future HSI despite sufficient convalescence. Additionally, a number of suggested post-HSI maladaptations are thought to contribute to the increased risk of future injury. These maladaptations include the formation of non-functional scar tissue(49) that is associated with an alteration in muscle tissue lengthening mechanics,(101) reduced flexibility,(44, 93, 95) persistent reductions in eccentric strength,(43-45, 102) long term atrophy of the injured muscle,(46) alterations in the angle of peak knee flexor torque(76) and alterations in lower limb biomechanics.(20) Given the retrospective nature of these observations(43-46, 49, 76, 93, 95, 101, 102) it is difficult to ascertain if these traits are the cause of or the result of previous injury, however it is accepted that modifications (or maladaptations) do occur following HSI.(49) From the available literature, persistent reductions in eccentric strength, (43-45, 102) the alterations in the angle of peak knee flexor torque(69, 76, 90) and reduced flexibility(44, 93, 95) have been examined most extensively in the literature and will be discussed in the following sections. The

emerging evidence relating to the impact of scar tissue on muscle tissue lengthening mechanics,(101) however, is also worthy of further discussion. Findings from Silder and colleagues(101) suggest that previous hamstring injury at the muscle-tendon junction results in a proliferation of scar tissue in this region and ultimately leads to adjacent muscle fibres experiencing greater strain during eccentric contraction. Such an adaptation to muscle tissue lengthening mechanics following injury would imply a greater risk of re-injury given the association between higher levels of muscle fibre strain and susceptibility to muscle damage.(62)

The high rate of recurrence and the elevated risk associated with previous injury highlights the importance of preventing first-time HSI and avoiding the vicious injury-reinjury cycle.(49) Furthermore, whilst previous injury has been identified as elevating the risk of future injury, much work still needs to be done to determine what maladaptations are responsible for this increased risk.

2.7.1.3 Ethnicity

Three independent studies have identified Aboriginal(20) and black African or Caribbean(9, 15) ethnicity as risk factors for HSI, however only one study reported the risk to be significantly increased (Odds ratio = 11.2; 95%CI: 2.1 to 62.5).(20) Both high proportions of type II fibres(78, 103) and excessive anterior pelvic tilt(15, 84) have been suggested as factors in the incidence of HSI in these populations, however, these are not substantiated and more objective evidence is required to determine how ethnicity impacts upon HSI risk.

2.7.2 ALTERABLE RISK FACTORS

2.7.2.1 Strength imbalances

Strength imbalances of the hamstring muscle group have long been suggested as causes of HSI.(87) For the purposes of this review a strength imbalance can include any of the following: knee flexor weakness, bilateral knee flexor strength asymmetry and low ratios of knee flexor to knee extensor strength, otherwise known as hamstring to quadriceps or H:Q ratios.

2.7.2.1.1 Strength

Experimental data from animal models has shown that fully stimulated muscles are able to withstand greater amounts of stress before stretch-induced failure compared with partially activated muscles.(58) The authors postulated that stronger muscles would provide greater protection from strain injury and that muscle weakness may be a risk factor for muscle strain injury,(58) however the evidence linking hamstring weakness to HSI in humans is mixed.(5, 88, 89) Whilst one prospective study has found that subsequently injured Australian footballers demonstrated lower peak concentric hamstring torque in preseason isokinetic testing,(88) this finding was not replicated in a larger but otherwise similar study a year later.(5) Prospective data on sprinters supports the findings of Orchard and colleagues(88) as isometric knee flexion strength relative to body weight was significantly lower in subsequently injured limbs.(89)

2.7.2.1.2 Bilateral asymmetry

Testing to assess unilateral hamstring strength allows for the determination of a weaker limb, if one exists. It has been proposed that a significantly weaker hamstring on one limb compared to the contralateral limb, termed hamstring bilateral asymmetry, may predispose the weaker hamstring to an elevated risk of injury.(104) The use of a between limb comparison of strength may be a more meaningful marker of weakness for individuals than a comparison with a group average or standardised score.

Early studies suggested that between limb hamstring strength asymmetry of greater than 10% was a predictor of hamstring injury in American footballers and track and field athletes.(47, 87) Later, elite Australian footballers with a bilateral asymmetry of 8% or more were found to have an increased risk of HSI(88) whilst soccer players with an asymmetry of more than 15% were at an increased risk.(32) It should be noted, however, that some authors have found no predictive power of bilateral strength imbalances.(5, 90)

Whilst some disagreement exists in the literature to date, a number of studies have identified that bilateral hamstring asymmetry leads to an increased risk of sustaining a HSI in a number of athletic cohorts.(32, 33, 47, 87, 88) Further exploration of imbalances between the hamstrings and other muscles of the hip joint is warranted as this may impact upon hamstring loading particularly during the terminal swing phase of running. Any alterations in running biomechanics associated with hamstring

strength asymmetry should also be explored to determine if hamstring loading is affected as a result of imbalance.

2.7.2.1.3 Hamstring: Quadriceps strength ratio

A lower H:Q ratio suggests a relatively poor capacity for the hamstrings to act as 'brakes' at the flexing hip and extending knee joints during the terminal swing phase of running. Thus forceful contraction of the quadriceps, as occurs during the early swing phase of gait, has the potential to produce angular momentum at the knee joint that exceeds the mechanical limits of the hamstring.(105) Initial research(47, 87, 88) focused on comparisons of concentric strength imbalances across the knee joint ($H:Q_{conv}$) but has been criticised as it neglects the functional role of the hamstrings during the terminal swing phase of gait, that of a forceful eccentric contraction.(34, 37, 38, 53) More recently the comparison of eccentric hamstring to concentric quadriceps strength, known as a functional strength ratio ($H:Q_{func}$), has been suggested(105) and popularised.(5, 32, 33, 43, 90)

One of the earliest studies to examine the relationship between $H:Q_{conv}$ ratios and future injury risk found that American footballers with a $H:Q_{conv}$ ratio of less than 0.50 were at an elevated risk of HSI.(47, 87) A later small-scale study in Australian footballers found that a $H:Q_{conv}$ of less than 0.61 put an individual at a substantially increased risk of HSI(88) whilst a larger study performed only a year later was unable to find an association between $H:Q_{conv}$ or $H:Q_{func}$ ratio and future HSI in Australian footballers.(5) These studies employed athletes at different levels of expertise and professionalism and employed different methodologies, all of which

make comparison of the findings difficult. With respect to sprinters, prospective observations found that neither $H:Q_{conv}$ or $H:Q_{funct}$ displayed any significant differences between athletes who did or did not suffer a HSI.(90) Whilst Cox regression analysis did determine that a $H:Q_{conv}$ ratio below 0.60 lead to an increase in the risk of sustaining a HSI by 17.4 (95%CI: 1.3-231.4) fold(90) the sample size of the injured group (*n*=8) should have precluded use of this statistical method. Other prospective observations have found that pre-season $H:Q_{funct}$(33) and an isometric H:Q(89) were significantly lower in the subsequently injured limbs of sprinters.

Many of these studies are limited due to their small sample sizes which makes detecting small associations between H:Q and HSI risk difficult.(106) The most powerful study to have examined the association between H:Q ratio and HSI (*n*=462) found that uncorrected strength imbalances in soccer players, which included a $H:Q_{conv}$ below 0.45-0.47 (exact cut-off depends on dynamometer brand used) and a $H:Q_{funct}$ below 0.80-0.89 were associated with a significantly greater frequency of HSI compared to athletes without strength imbalances.(32) Furthermore, the correction of strength imbalances, including normalising H:Q ratios, led to a significant reduction in HSI frequency compared to athletes with uncorrected imbalances (see Chapter 1.7.3.2).(32) These findings provide the strongest evidence available that sufficient H: Q ratios protect athletes from future HSI.

2.7.2.1.4 Angle of peak knee flexion torque

Athletes with a greater knee angle at peak concentric knee flexion torque (those who produce peak knee flexor torque at shorter muscle lengths) are proposed to be at greater risk of HSI.(76) The hamstrings in these individuals would be expected to work on the descending limb of the length-tension relationship across a greater range of motion, leaving them more prone to damage.(107)

Athletes with a history of unilateral hamstring injury display peak knee flexion torque at a greater degree of knee flexion on their injured limb compared to the uninjured limb (Figure 2-1),(76) however, it is not known if this is the cause of or the result of previous injury given the retrospective nature of these observations. In an attempt to determine a relationship between the angle of peak torque and future HSI occurrence, a recent prospective study in elite and sub-elite Japanese sprinters was performed.(90) This investigation found no association between the angle of peak knee flexor torque and subsequent HSI during the competitive season.(90) Currently the evidence pertaining to the usefulness of the angle of peak knee flexor torque to predict previous or future HSI is too sparse to draw any firm inferences and more work in this area is required.

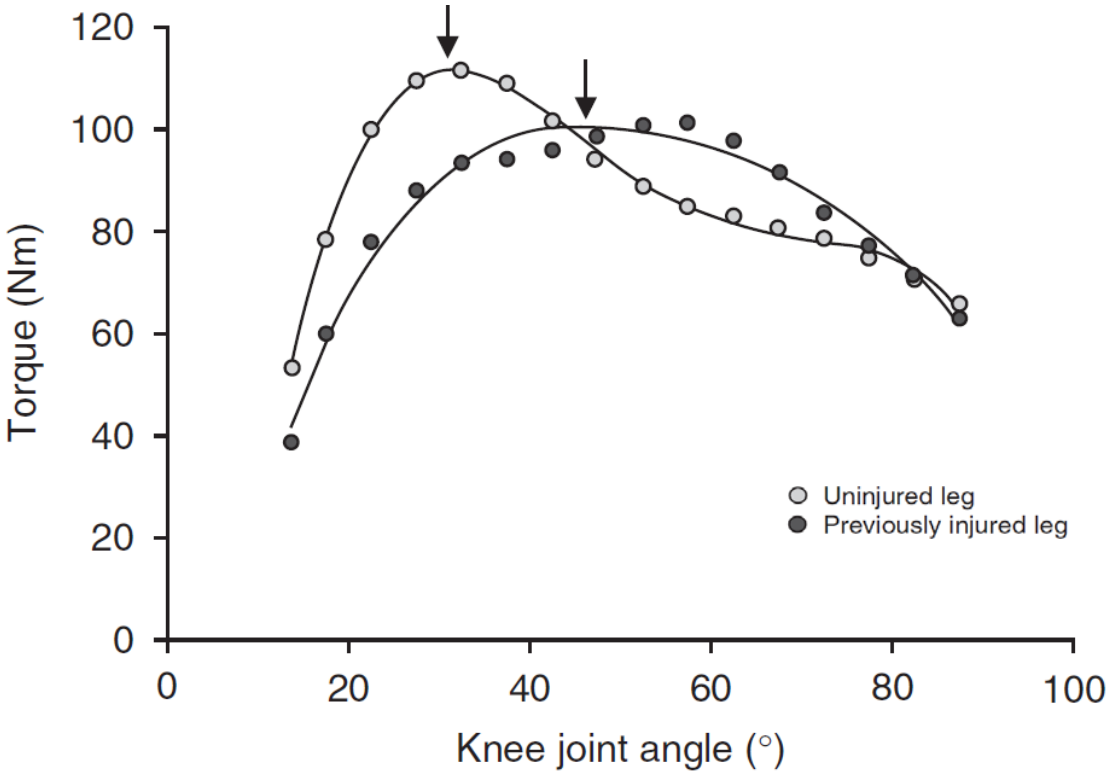

Figure 2-2. Unpublished concentric knee flexor torque-joint angle relationships from a single elite male athlete tested at $60^{o}.s^{-1}$ in our laboratory. Angle of peak torque is indicated by the downward arrows. 0° indicates full knee extension, 100° indicates 100° of knee flexion. The previously injured hamstring produces its peak torque at shorter muscle lengths (greater angle of peak torque), and hence operates to a greater extent along the descending limb of the length-tension curve.

2.7.2.2 Flexibility

Flexibility training has traditionally been proposed as a key component of injury prevention in athletes despite a lack of convincing prospective scientific evidence.(97, 108) It is proposed that greater flexibility may reduce the risk of strain injury due to a greater ability of the passive components of the muscle-tendon unit to absorb energy as a result of greater compliance(108, 109) although this point is disputed in the literature.(110)

Prospective studies in both American(87) and Australian footballers(6, 88, 109) have found no relationship between the hamstring flexibility from the sit-and-reach or toe-touch test and future HSI risk. In contrast to popular belief, Australian footballers with a history of HSI, who displayed greater sit-and-reach flexibility were actually more likely to sustain a recurrent HSI.(6) Furthermore, poor hamstring flexibility, as assessed via an active or passive knee extension test or a straight leg raise, did not increase the risk of HSI in Australian footballers,(111) soccer players(42) or sprinters.(90) In contrast, some studies have reported relationships between flexibility and hamstring injury.(16, 92, 94) A study in elite soccer players found that hamstring flexibility of less than 90 degrees in a passive straight leg raise correlated significantly with future HSI.(94) Further studies also identified reduced hamstring flexibility as a significant independent risk factor for HSI in elite soccer players.(16, 92)

Whilst the weight of evidence suggests that there is no protective benefit of greater hamstring flexibility on HSI risk, methodological flaws exist with the measurement techniques employed. Foreman and colleagues(112) suggest that no gold standard measurement for flexibility has been established and that tests of hamstring length such as the sit and reach, straight leg-raise and toe touch test can be inaccurate if they do not allow for stabilisation at the hip and lumbar spine.

Future studies should employ more objective measures of flexibility such as the method used by Arnason and colleagues(42) which involves a tension meter to determine the limits of range of motion. This is as opposed to the subjective assessment of the end of range of motion by the investigator or subject which may

display good levels of inter- and intra-tester reliability but may suffer with respect to ecological validity. Even if such subjective measurements are reproducible there is no means of determining whether a subject has been stretched to their maximal range of motion. The use of a more objective approach would be expected to improve the ecological validity of clinical flexibility tests given that a set level of passive tension is defined as the end of range for all subjects.

2.7.2.3 Fatigue

Fatigue and its associated performance decrements have often been suggested as causative factors for injury.(47, 59, 97) Indeed, studies of injury incidence have shown that HSIs occur at a greater rate in the latter stages of competitive matches and training.(9, 13, 15, 113, 114)

The effect of fatigue on muscle lengthening properties was initially examined in a laboratory setting. In these experiments, muscles that were pre-fatigued via electrical stimulation absorbed less energy before failure when compared with unfatigued muscles.(59) Fatigued and control muscles still failed at the same length, indicating that a fatigued muscle may be more likely to suffer a strain injury due to a reduced capacity to resist over-lengthening.(59) With respect to human muscle function, one group has shown that fatigue of the hamstrings induced by repeated dynamic efforts leads to an increase in the amount of knee extension observed during the terminal swing phase of running.(115) This increase in knee extension would be expected to lead to a greater strain on the hamstrings during the terminal swing phase of gait,(37) however, it was matched by a reduction in hip flexion.(115) These alterations in knee

and hip joint positions suggest that fatigue from dynamic exercise may lead to alterations in proprioception, a phenomenon which has been reported in response to other experimental models of knee flexor fatigue.(116) In these trials, isokinetic exercise which induced a 30% reduction in knee flexor maximum voluntary contraction force resulted in a reduction in proprioceptive ability, whereby hamstring length was under-estimated in a fatigued state.(116) This could lead to the perception of normal hamstring muscle lengths during running whilst in reality repeated over-lengthening of the hamstrings is occurring. Such deficits in proprioception when fatigued may elevate the risk of HSI given the assertions made by Morgan(69) that continual over-lengthening would lead to microscopic muscle damage that may accumulate to become macroscopic damage (i.e. strain injury).

More recent work has also shown that intermittent running designed to mimic the demands of competitive soccer significantly reduces eccentric hamstring torque with little or no impact on concentric knee flexion or extension strength.(39, 117) In our own unpublished work we have found marked variability in the loss of eccentric hamstring strength. Those who exhibit greater levels of preferential eccentric hamstring fatigue would be expected to be at a greater risk of a HSI with prolonged activity given the link between eccentric weakness and HSI risk.(32, 33)

Other potential factors linking fatigue with elevated risk of muscle strain injuries, such as altered technique, reductions in concentration and other intrinsic physiological changes such as reduced coordination of muscle recruitment patterns, have been suggested(96) but are yet to be rigorously tested.

2.7.3 ADDRESSING RISK FACTORS TO REDUCE THE RISK OF HAMSTRING STRAIN INJURIES

Intervention studies and randomised control trials are important in determining if reported risk factors are indeed causative factors in injury aetiology. These study designs can determine whether interventions intended to improve purported causative factors result in reductions in the risk of sustaining a HSI. In fact risk factors cannot be considered causative unless there is a reduction in the risk of sustaining a HSI following an intervention aimed at ameliorating them.

2.7.3.1 Eccentric strength training

2.7.3.1.1 Nordic hamstring exercise

Two randomised control trials(118, 119) and one intervention study(65) have examined the benefits of Nordic hamstring exercise (NHE) on HSI rates. The NHE is a body weight exercise that requires athletes to begin in a kneeling position and to gradually lower their upper bodies towards the ground by extending at the knee while contracting the knee flexors eccentrically to slow the descent. During the exercise the athlete's ankles are typically held down by a partner.(120) The NHE has been shown to increase eccentric hamstring torque(120) and shift the torque-joint angle curve of the hamstrings to longer muscle lengths(121) and both are suggested mechanisms by which the NHE may reduce HSI rates.

The implementation of NHEs failed to reduce rates of HSI in cohorts of amateur Australian footballers(119) and professional soccer players,(118) however compliance with both intervention programs was extremely low. Gabbe and colleagues(119) reported that approximately half of all participants allocated to their

intervention group did not complete the second training session and that fewer than 10% completed the five planned sessions. Engebretsen and colleagues(118) also reported that only 21% of players performed 20 or more of 30 planned sessions of NHEs. Furthermore, the use of extremely high volume and low frequency (once per two to three weeks) hamstring training in one of these interventions(119) was inconsistent with conventional conditioning practices.(120)

In contrast, elite soccer teams who chose to implement the NHE as part of their pre-season and in-season conditioning programs displayed a 65% reduction in HSI compared to teams that did not.(65) Furthermore, the teams that utilised the intervention displayed significantly lower rates and severity of HSI compared with previous seasons.(65) This study was, however, limited by a non-randomised approach as individual teams decided if they were to participate in the intervention. Interestingly the implementation of NHEs did not reduce the rate of HSI recurrence.(65)

2.7.3.1.2 Flywheel training

Training on a flywheel ergometer(122) which is designed to augment the amount of eccentric torque required during the performance of a lying leg curl, has been reported to increase eccentric hamstring strength and reduce HSI rates.(61) A small scale randomised control trial performed on two elite soccer teams (n=30 players) found that flywheel hamstring training in the pre-season significantly reduced the number of HSI compared to the control group. However, the control group displayed a remarkably high rate of HSI incidence (66%)(61) and this potentially diminishes the significance of these findings.(123)

2.7.3.1.3 Considerations for exercise selection

At present the literature pertaining to the benefits of eccentric strength training on reducing HSI incidence is inconclusive.(123) Whilst a number of factors, including a lack of compliance to eccentric strength training interventions,(118, 119) may contribute to this, exercise selection may also be a factor. The SM and ST reportedly exhibit greater activation levels at shorter muscle lengths, whereas the BF_L is most powerfully activated at longer lengths during isokinetic knee flexion.(124)

Magnetic resonance imaging has recently revealed that the BF_L and SM muscles were significantly less active than the ST and gracilis during a heavily loaded eccentric leg curl, which mimics the knee range of motion and hamstring lengths experienced in the NHE and flywheel training.(125) It is therefore possible that these exercises may be sub-optimal in bringing about adaptation in the BF_L, the muscle most frequently injured.(22-24) Exercises that better target the BF_L such as the stiff-legged deadlift(126) may prove more effective in hamstring injury prevention than those that have so far been employed in randomised control trials.

2.7.3.2 Strength imbalance correction

A large scale cohort study (*n*=462) of isokinetic hamstring strength in elite soccer players found that correction of strength deficits (either concentric or eccentric asymmetries or low H:Q ratios) lead to similar HSI rates compared to athletes without strength deficits.(32) Participants who had strength deficits but did not undergo isokinetic rehabilitation or who did undergo isokinetic rehabilitation but did not perform post-intervention testing showed significantly higher rates of HSI.(32)

This study is of great significance as it employed one of the largest sample sizes of any HSI prevention study and suggests that a reduction in the risk of HSI can be achieved via the detection and subsequent correction of isokinetic strength deficits.

2.7.3.3 Flexibility training

An intervention study performed on elite soccer players found that a prescribed contract-relax flexibility training protocol performed during the warm up did not reduce the rate of HSI compared with teams that did not incorporate flexibility training.(65) Similarly a randomised control trial involving recreational level runners, who completed a 16-week unsupervised intervention consisting of warm-up and cool-down procedures and stretching showed no difference in the rate of HSI compared to a control group.(127) Consideration must, however, be given to the potential that the intervention may have been inadequate to increase flexibility due to the brief duration of stretching exercises (10 seconds).(123) These finding are not totally unexpected given the lack of evidence for poor flexibility being a risk factor for HSI (see Chapter 2.7.2.2). However further work needs to be performed, with greater control over other confounding variables such as aerobic and eccentric hamstring conditioning to fully elucidate the effect of flexibility training on HSI rates.

2.8 HAMSTRING STRAIN RECURRENCES AND NEUROMUSCULAR INHIBITION

Whilst there is an extensive list of risk factors for HSI which have been examined through a number of different methodological designs, epidemiological data suggest that first-time and recurrent HSI rates in sport are not in decline.(1, 2, 7, 9, 13, 15, 29) This suggests that our current understanding of what increases the risk of a future HSI has not accounted for all contributing factors or that we are unable to resolve previously identified factors effectively. Despite previous HSI being consistently identified as one of the primary risk factors for a future HSI(6, 20, 41, 42, 86) maladaptation associated with HSI, particularly nervous system function, has been largely overlooked. Potentially, a number of reported maladaptations associated with prior HSI may be explained by a common neurological mechanism in response to previous injury.

Weakness after painful musculoskeletal injury is typically mediated by both muscular and neural adaptations. For example, following traumatic knee injuries involving anterior cruciate ligament (ACL) ruptures maximal voluntary activation of the quadriceps is significantly reduced, even years after the injury occurred(128, 129) and despite restoration of knee stability.(129) In the case of HSI, however, little attention has been paid to the possibility that prolonged deficits in activation contribute to the high injury recurrence rate. This is surprising given that the torque-velocity relationships of previously injured hamstrings are characteristic of heightened neuromuscular inhibition in the sense that they show greater deficits in eccentric than concentric strength.(44, 45, 102) Prolonged neuromuscular inhibition at long muscle lengths after HSI could potentially account for observations of

preferentially eccentric weakness,(44, 45, 102) persistent atrophy of the previously injured muscles(46) and alterations in the angle of peak knee flexor torque,(76) all of which are purported risk factors for HSI and have been observed in athletes following 'successful' rehabilitation and the return to full competition and training.

A reduction in the capacity of the nervous system to activate injured muscles presumably constitutes a strategy to unload damaged tissues and thereby reduce pain in the acute recovery period. As the greatest pain after HSI is typically felt at longer muscle lengths, it is not surprising that there is now evidence for a length-specific reduction in hamstring activation.(130) Inhibition, particularly during eccentric actions and at longer muscle lengths, may also impede the rehabilitation process by limiting adaptations within the previously injured muscle(s).

The early and middle stages of treatment for HSI are characterised by the avoidance of excessive stretching to prevent further scar formation and submaximal exercises performed through a limited range of motion and with hip joint movements restrained primarily to the frontal plane.(131) Thus, by the time athletes are in the late stages of rehabilitation, their hamstring muscles might be expected to have shed in-series sarcomeres(132) and to have atrophied considerably. Having fewer in-series sarcomeres would be expected to shift the peak of the knee flexor torque-joint angle curve to shorter muscle lengths and create even greater weakness at longer lengths than atrophy alone.(76) Such hamstring function is detrimental as running requires strength at relatively long muscle lengths to decelerate hip flexion and knee extension during terminal swing.(34, 36, 37)

The return to running at progressively faster speeds and the use of more intense strengthening exercises later in rehabilitation should increase exposure to forceful eccentric actions at relatively long muscle lengths(34, 36-38, 54) and might therefore be expected to return muscles to their original size and fascicles to their pre-injury lengths.(133) However, any lingering neuromuscular inhibition would spare the previously injured hamstring muscle(s) from significant activation during eccentric actions at long length and would therefore limit or prevent hypertrophy and sarcomerogenesis. Evidence of persistent atrophy in the previously injured BF_L with simultaneous compensatory hypertrophy of the uninjured BF_S in recreational level athletes, 5-23 months after HSI and after a full return to training and competition(46) is consistent with the hypothesis of prolonged muscle-specific inhibition.

Additional investigation is required to confirm whether previously injured athletes display significantly greater levels of neuromuscular inhibition within the previously injured leg compared to their contralateral uninjured limb and whether inhibition is confined specifically to the injured muscle. Ultimately to identify neuromuscular inhibition as a causative factor in recurrent HSI, prospective studies and randomised control trials need to be performed to determine if inhibition following HSI results in an increased risk of re-injury and whether ameliorating this neurological deficit reduces the incidence of recurrent HSI. Techniques such as electromyography (EMG),(124, 130, 134-136) twitch interpolation(137, 138) and electrical stimulation(135, 139, 140) have been used previously to assess voluntary muscle activation and all should be considered for future work in this area. Further work also needs to be carried out to rigorously determine the full extent of physiological maladaptation associated with altered neural function following HSI.

2.9 CONCLUSION

HSIs remain the predominate injury in a number of sports despite concerted efforts to expand scientific knowledge. Additionally, HSI have shown a high rate of recurrence and the capacity to impact negatively on individual and team performance and financial viability of elite sports clubs. Whilst it is widely acknowledged that the causes of HSI are multifactoral, the interaction between these factors is often overlooked. This review has integrated the role of the hamstrings in running, the specifics of hamstring anatomy and reported risk factors and interventions for HSI to better understand the causes of this injury.

Sports medicine practitioners and sports injury researchers alike need to appreciate the complex nature of HSI and understand that no one single approach can be considered the gold standard for HSI prevention or rehabilitation. For example, a focus solely on markers of performance (i.e. eccentric strength, flexibility) may neglect the important role that correct running technique may have on injury avoidance. The biomechanical demands of running, the anatomical organisation of the hamstrings and a range of unalterable and alterable risk factors, such as age, previous injury, ethnicity, strength imbalances, flexibility and fatigue have all been linked to HSI. All of these factors need to be considered, as does the interaction between these factors and the impact of reported interventions, by practitioners looking to prevent HSI. Furthermore, understanding of the exact causes of HSI remains elusive but muscle strain, high force eccentric contraction, accumulated muscle damage and/or a single injurious event may all potentially play a role and all should be considering when developing HSI preventative strategies.

Further to this, more work needs to be carried out in the area of assessing maladaptation associated with previous HSI. Whilst it is commonly known that previous HSI is the primary risk factor for future injury very little is known about the maladaptations associated with a previous insult. Understanding only that previous injury elevates the risk of injury without an understanding as to why, gives little insight into how HSI should be successfully rehabilitated. We propose a novel integrated framework of how previous injury may lead to persistent neuromuscular inhibition which could conceivably result in a cascade of maladaptations that elevate the risk of future HSI. This area should be a focus of future research given the high levels of HSI recurrence for a number of years in many sports.

3 STUDY ONE

Knee flexor angle of peak torque does not influence the amount of knee flexor strength lost following soccer specific intermittent running.

This chapter comprises the following manuscript currently under review in European Journal of Applied Physiology:

Opar DA, Williams MD, Porter KN, Raj IS, Shield AJ. Knee flexor angle of peak torque does not influence the amount of knee flexor strength lost following soccer specific intermittent running. European Journal of Applied Physiology. In review.

The authors listed have certified that:

1. they meet the criteria for authorship in that they have participated in the conception, execution, or interpretation, of at least that part of the publication in their field of expertise;
2. they take public responsibility for their part of the publication, except for the responsible author who accepts overall responsibility for the publication;
3. there are no other authors of the publication according to these criteria;
4. potential conflicts of interest have been disclosed to (a) granting bodies, (b) the editor or publisher of journals or other publications, and (c) the head of the responsible academic unit, and
5. they agree to the use of the publication in the student's thesis and its publication on the QUT ePrints database consistent with any limitations set by publisher requirements.

In the case of this chapter, the following contributions were made:

Contributor	Statement of contribution
David Opar	Determined experimental design, gained ethical approval, participant recruitment, data collection, data analysis, statistical analysis, wrote and submitted the manuscript. Signature: DO Date: 13/5/13
Morgan Williams	Determined experimental design, data analysis, statistical analysis, assisted writing the manuscript.
Kirsten Porter	Participant recruitment, data collection, data analysis.
Isaac Selva Raj	Determined experimental design, participant recruitment, data collection, data analysis.
Anthony Shield	Determined experimental design, assisted with ethical approval, data analysis, statistical analysis, assisted writing the manuscript.

Principal Supervisor Confirmation

I have sighted email or other correspondence from all Co-authors confirming their certifying authorship.

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Name Signature Date

3.1 LINKING PARAGRAPH

As outlined in Chapter 2.7.2.3, fatigue appears to be implicated in the aetiology of hamstring strain injury (HSI). The relationship between prolonged time within a half of rugby union and soccer and greater incidence of HSI is most likely mediated by the reported declines in eccentric hamstring strength following repeat sprint and/or intermittent running protocols designed to mimic the demands of running based sports. This lessening of eccentric hamstring strength would be expected to increase the likelihood of HSI as lower levels of eccentric hamstring strength have been identified as risk factor for future injury. The mechanism responsible for this eccentric specific decline in hamstring function following running remains unknown, however, and is the focus of Chapter 3. It is hypothesised that the knee flexor angle of peak torque may distinguish between individuals who show greater and lesser declines in eccentric knee flexor strength from, as per the sarcomere popping hypothesis (Chapter 2.5).

3.2 OVERVIEW

The purpose of this study was to determine whether knee flexor angle of peak torque influenced the extent of knee flexor strength lost following a soccer specific treadmill running protocol. Fifteen recreationally active males completed concentric and eccentric isokinetic strength tests of the knee flexors and a concentric strength test of the knee extensors prior to, immediately after and 15 minutes after a 45-minute intermittent treadmill run. Knee flexor angle of peak torque was determined from isokinetic data. Treadmill running caused a 15.0 ± 7.9% decline in eccentric knee flexor strength ($p<0.001$) without altering concentric knee flexor or extensor torque. There were two distinct distributions in eccentric knee flexor strength loss and the reduction in the functional hamstrings to quadriceps ratio ($H:Q_{func}$), suggesting the existence of high and low responders to intermittent running. Knee flexor angle of peak torque ($p=0.937$) did not differ between high and low responders, although high responders were weaker in concentric tests of knee flexor strength at $60^{0}\cdot s^{-1}$ ($p=0.016$) and tests at $180^{0}\cdot s^{-1}$ when collapsed across time ($p=0.009$). Knee flexor angle of peak torque does not influence the extent of eccentric knee flexor weakness after intermittent treadmill running. The impact of intermittent running on eccentric hamstring fatigability is highly variable and further investigation is required to determine what factors influence the amount of knee flexor strength lost following intermittent running.

3.3 INTRODUCTION

HSIs are prevalent in sports that involve intermittent and repeat sprint running such as Australian football (7), rugby union(9) and soccer(15). Most believe that the terminal swing phase of the running cycle is most injurious (141, 142), given the high force eccentric contraction required to decelerate the flexing hip and extending knee.(37, 38) Given the eccentric demands placed on the hamstrings during running it is not surprising that the most compelling evidence suggests that increasing eccentric knee flexor strength is the most effective method to reduce the risk of HSI.(61, 143) Not surprisingly, therefore, eccentric knee flexor weakness is a known risk factor hamstring strain injury (HSI).(33) Interestingly, fatigue appears to be implicated in the development of HSIs as a some studies report more injuries in the later portions of each half in rugby union and soccer matches.(9, 15) The role of fatigue in HSIs may be mediated by the fact that following intermittent and repeat sprint running, declines in knee flexor function has been largely confined to reductions in eccentric strength and the associated $H:Q_{func}$.(39) In contrast, concentric function remains largely unchanged following these running protocols.(39) The determination of the mechanisms responsible for this eccentric specific decline in function is paramount if effective interventions are to be implemented to lessen the decline in eccentric knee flexor strength and thus reduce the risk of HSI with prolonged activity. The eccentric specific nature of this phenomona suggests that the mechanism responsible for these declines in function must be unique to eccentric contraction. It is well established that accumulated microscopic muscle damage is seen following eccentric, but not concentric contraction,(144) making eccentrically induced muscle damage a potential

mechanism which influnces eccentric knee flexor strength following intermittent or repeat sprint running.

One way to determine if accumulated microscopic muscle damage does impact upon the decline in eccentric knee flexor strength following running is to examine whether those who are most prone to knee flexor damage are also those who show the greatest decline in eccentric strength and the $H:Q_{func}$ following a running protocol. Particularly for the knee flexors, a concentric, isokinetically dervied measure (typically at $60^{o}.s^{-1}$) of the knee angle of at which peak torque occurs (the angle of peak torque) has been popularised as a technique to determine the propensity for muscle damage.(121) It has been proposed that knee flexor muscles which reach peak torque further from full knee extension, thereby working further along the descending limb of their length-tension relationships, will experience greater damage and weakness during and after running (76, 145). However the impact of differing knee flexor angles of peak torque on the extent of knee flexor weakness following intermittent running has not yet been examined. We therefore tested the hypothesis that subjects who exhibited peak torque at greater angles of knee flexion (shorter muscle lengths) would lose the most knee flexor strength and show the greatest decline in the associated $H:Q_{func}$ ratio after a soccer specific treadmill running protocol. Because of a serendipitous finding of two distinctly different patterns of eccentric force loss in our participants, we were able to compare angle of peak torque and pre-exercise strength levels between subjects who lost large and small amounts of eccentric knee flexion strength after treadmill running.

3.4 METHODS

3.4.1 PARTICIPANTS

Fifteen physically active males volunteered to participate in the study. All participated in running based sports (i.e. Australian football, athletics, soccer) and were free of any lower limb injury and other conditions that might be expected to impact running gait or isokinetic strength tests. Written informed consent was provided by all participants and the study was approved by the University Human Research Ethics Committee. After familiarisation with the experimental procedures and the performance of a treadmill test of maximal oxygen consumption, all participants performed unilateral isokinetic tests of knee flexor and extensor strength before (Pre), immediately after (Post) and 15 minutes after ($Post_{15}$) a 45 minute intermittent treadmill running protocol which replicated the physiological demands of a half of soccer (146).

3.4.2 PEAK VOLUME OF OXYGEN UPTAKE

At least three days prior to strength testing, participants completed a peak volume of oxygen uptake test using a previously described protocol (147). The test, administered on a motorised-treadmill (Pulsar, HP Cosmos, Nussfort-Traunstein, Germany), began at 8.5 $km \cdot hr^{-1}$, with the speed increasing by 1 $km \cdot hr^{-1}$ every minute until 14.5 $km \cdot hr^{-1}$ was attained. The gradient was then increased by 2% every minute until volitional fatigue, despite continual encouragement from the investigators.

3.4.3 INTERMITTENT RUNNING PROTOCOL

The soccer-specific treadmill protocol used in this study was based on one described previously (146). It involved a range of running velocities (from 4 to 25 $km \cdot h^{-1}$) and occasional periods of standing still with frequent transitions from one velocity to another. In contrast to the original protocol(146) that involved two 45 minute 'halves', participants in this study ran 4.86 km in a single 45 minute period to simulate a single half of competitive soccer.

3.4.4 ISOKINETIC DYNAMOMETRY

Participants completed two familiarisation sessions on a Biodex System 4 isokinetic dynamometer, with the final familiarisation session at least six days prior to the full experimental protocol. Isokinetic tests were limited to the dominant limb, defined as the preferred kicking leg. Contractions of the knee flexors and extensors were performed between the angles of 100^{o} and 10^{o} of knee flexion (full extension = 0^{o}) with participants in a seated position. Hip angle was flexed approximately 85^{o} from the anatomical position and participants were restrained with straps across the hips, active thigh and trunk. Correction for limb weight was performed. Before pre-exercise isokinetic tests, participants performed a warm-up consisting of two sets of four submaximal concentric contractions of the knee extensors and flexors at an angular velocity of $35^{o}.s^{-1}$. They then performed two sets of four consecutive maximal contractions involving concentric knee extension and flexion at $60^{o}.s^{-1}$ with one minute of rest between sets. Thereafter, participants completed two sets of four maximal concentric contractions for knee extensors and flexors and two sets of four maximal eccentric knee flexor contractions at $180^{o}.s^{-1}$ with 30 seconds between sets.

The order of concentric and eccentric tests at this higher velocity was randomised. During eccentric knee flexion contractions the dynamometer was returned to the starting position for the next contraction by one of the experimenters. Participants repeated the concentric knee extension and flexion and eccentric knee flexion contractions at $180^{o}.s^{-1}$ immediately after and 15 minutes after the cessation of running to assess any alterations in strength, as per previous protocols. Participants remained seated on the dynamometer between post-exercise tests.

3.4.5 DATA ANALYSIS

Dynamometer torque and angle data were transferred to computer at 1 kHz and stored for later analysis. Peak torque for the knee flexors and extensors was defined as the mean maximal torque of the eight contractions for each contraction modes and velocities. The $H:Q_{func}$ was determined as the quotient of eccentric knee flexion torque at $180^{o}.s^{-1}$ and concentric knee extension torque at $180^{o}.s^{-1}$. Torque-joint angle relationships for the knee flexors were constructed from the concentric contraction data collected a $60^{o}.s^{-1}$. For each contraction, torques were averaged across 5^{o} range of motion bins between 15^{o} and 95^{o} of knee flexion and across one 2.5^{o} bin between 12.5^{o} and 15^{o} of knee flexion. Angle-specific torques from all eight contractions were then averaged together. Fourth order polynomial functions were fitted to the mean torques and joint angles of each bin using OriginPro 8.1 software (OriginLab, Northampton, MA). Joint angles of peak torque were then determined from the peaks of the fitted curves.

3.4.6 STATISTICAL ANALYSIS

We originally intended to examine the correlation between angle of peak torque measured at $60^{o}.s^{-1}$ and the decline in eccentric knee flexor torque measured at $180^{o}.s^{-1}$ via bivariate parametric means, however, the observed decline in eccentric strength was bi-modally distributed. Seven participants exhibited 20% or more of a decline in eccentric knee flexor torque (high responders) while eight others exhibited smaller losses of between 5 and 12% (low responders). The distribution of eccentric strength loss was normal within these groups. Consequently, we compared the responses for high and low responders (Group) by time (Pre, Post, $Post_{15}$) using a restricted maximum likelihood method for fitting mixed models for each variable (concentric knee extension and flexion and eccentric knee flexion torque measured at $180^{o}.s^{-1}$ and $H:Q_{func}$) using JMP version 8.01 Statistical Discovery Software (SAS Inc). When main effects or interactions were detected, Tuckey's honestly significant difference post-hoc tests were used to identify the source of the significant differences. The least mean squares differences were reported with 95% confidence intervals (95%CI). Anthropometric characteristics, peak volume of oxygen uptake, pre-exercise concentric knee flexion and extension torque measured at $60^{o}.s^{-1}$ and the percentage decline in eccentric knee flexor torque measured at $180^{o}.s^{-1}$ for high and low responders were also compared using *t* tests. Knee flexor angle of peak torque was not normally distributed amongst high responders so the Mann-Whitney U test was employed to compare high and low responders. The test for knee flexor angle of peak torque was one-tailed as dictated by the hypothesis. Significance was set at 0.05 and the study had adequate power (0.8) to detect differences between high and low

responder groups of 6^{o} in angle of peak torque. Furthermore, to assess the magnitudes of the differences Cohen's d was calculated to report effect size (ES).

3.5 RESULTS

3.5.1 PARTICPANTS

Anthropometric and selected pre-exercise performance characteristics are shown in Table 3-1.

3.5.2 STRENGTH POST INTERMITTENT RUNNING

With respect to the immediate post-exercise decline in eccentric knee flexor torque measured at $180^{o}.s^{-1}$, there were two distinct distribution patterns of response, with seven high responders and eight low responders identified. Knee flexor angle of peak torque was not higher in high than low responders (low – high responders = 1.47^{o}; 95%CI = -6.42 to 3.48^{o}; p = 0.937; ES = 0.40), interestingly however, concentric knee flexor torque measured at $60^{o}.s^{-1}$ was significantly higher in those who lost relatively little eccentric strength after running (low – high responders = 27.9 Nm; 95%CI = 6.2 to 49.9 Nm; p = 0.016; ES =1.43) (Table 3-1). Figure 3-1 shows the impact of treadmill running on the strength measures taken at the three time points. Considering average responses of the entire participant pool, there was a significant decline in eccentric knee flexor torque measured at $180^{o}.s^{-1}$ after running (pre – post = 26.3 Nm; 95%CI = 20.9 to 31.6 Nm; p < 0.001; ES = 0.87), without significant changes in concentric hamstring (p = 0.294; ES = 0.11) or concentric quadriceps torque (p = 0.582; ES = -0.04) measured at $180^{o}.s^{-1}$. The reduction in the $H:Q_{func}$ after running for the entire participant pool is depicted in Figure 2.2 ($H:Q_{func}$ pre – post difference = 0.16; 95%CI = 0.11 to 0.21; p < 0.001; ES = 1.06). Neither eccentric knee flexor torque measured at $180^{o}.s^{-1}$ (post – $post_{15}$ difference = 2.1 Nm; 95%CI = -3.2 to 7.5 Nm; p = 0.582; ES = 0.07) nor the $H:Q_{func}$ (post – post15

difference = 0.00; 95%CI = -0.05 to 0.05; p = 0.993; ES = -0.01) changed further in the 15 minute recovery period.

3.5.3 HIGH VS. LOW RESPONDERS TO INTERMITTENT RUNNING

Significant group by time interactions were found only for eccentric knee flexor torque measured at $180^{o}.s^{-1}$ ($p < 0.001$) and the $H:Q_{func}$ ($p = 0.004$). Eccentric knee flexor torque declined after running in both groups (low responders pre-post = 15.7 Nm; 95%CI = 6.7 to 24.7 Nm; $p < 0.001$; ES = 0.68; high responders pre-post = 36.8 Nm; 95%CI = 27.2 to 46.4 Nm; $p < 0.001$; ES = 1.55) as depicted in Figure 3-1. Between group comparisons revealed no statistically significant difference in pre-exercise values of eccentric knee flexor torque measured at $180^{o}.s^{-1}$ (low – high responders = 24.2 Nm; 95%CI = -16.7 to 65.2 Nm; $p = 0.420$; ES = 0.93). Immediately after running, however, low responders exhibited greater eccentric knee flexor torque measured at $180^{o}.s^{-1}$ (low – high responders = 45.4 Nm; 95%CI = 4.4 to 86.4 Nm; $p = 0.026$; ES = 2.12) and this difference was maintained after fifteen minutes of recovery (low – high responders = 45.5 Nm; 95%CI = 4.6 to 86.5 Nm; $p = 0.026$; ES =1.80). Post-exercise $H:Q_{func}$ for each group were lower than the corresponding pre-exercise values (low responders pre-post = 0.09; 95%CI = 0.01 to 0.18; $p = 0.027$; ES = 0.72; high responders pre-post = 0.23; 95%CI = 0.14 to 0.32; $p < 0.001$; ES = 2.19), as shown in Figure 3-2. This ratio stabilised during the 15 minute recovery and no further significant changes for either high or low responder groups were observed. No significant group difference in $H:Q_{func}$ were found prior to treadmill running (0.10; 95%CI = -0.13 to 0.32; $p = 0.764$; ES = 0.64), but low responders demonstrated a higher $H:Q_{func}$ immediately after running than high

responders (low – high responders = 0.23; 95%CI = 0.01 to 0.46; p = 0.042; ES = 2.49). Fifteen minutes after exercise the difference between groups was not statistically significant, despite being similar in magnitude to that found immediately after completion of the treadmill protocol ($H:Q_{func}$ mean difference = 0.21; 95%CI = -0.01 to 0.44; p = 0.070; ES = 1.35). Concentric knee flexor strength measured at $180^{o}.s^{-1}$ did not change significantly after exercise and was higher in low compared to high responders when collapsed across time (low – high responders mean difference = 23.7 Nm; 95%CI = 7.2 to 40.2; p = 0.009).

Table 3-1. Anthropometric characteristics and selected pre-exercise performance measures for all participants and for high and low responders separately.

	All subjects	High	Low	p
n	15	7	8	
Age (years)	21.6 ± 3.1	22.4 ± 3.5	20.9 ± 2.7	0.350
Body mass (kg)	76.4 ± 7.3	73.9 ± 13.2	78.1 ± 12.2	0.530
Height (cm)	176.5 ± 7.3	173.7 ± 7.2	178.0 ± 8.6	0.315
Decline in eccentric knee flexor torque^ (%)	15.0 ± 7.9	22.7 ± 1.9	8.2 ± 2.7	< 0.001*
Peak volume of oxygen uptake ($ml \cdot min^{-1} \cdot kg^{-1}$)	56.7 ± 4.4	56.4 ± 6.1	56.9 ± 5.5	0.409
Angle of peak torque# (^{o})	32.3 (31.7-35.2)	32.1 (30.5-32.3)	34.5 (33.3-36.3)	0.929
Concentric knee flexor torque# (Nm)	138.1 ± 23.7	123.2 ± 21.2	151.2 ± 18.0	0.016*
Concentric knee extension torque# (Nm)	220.1 ± 33.1	209.2 ± 37.1	229.6 ± 28.1	0.249

*significant difference between high and low responders; ^measured at $180^o.s^{-1}$; # measured at $60^o.s^{-1}$. Data are means ± standard deviations or medians (inter-quartile range).

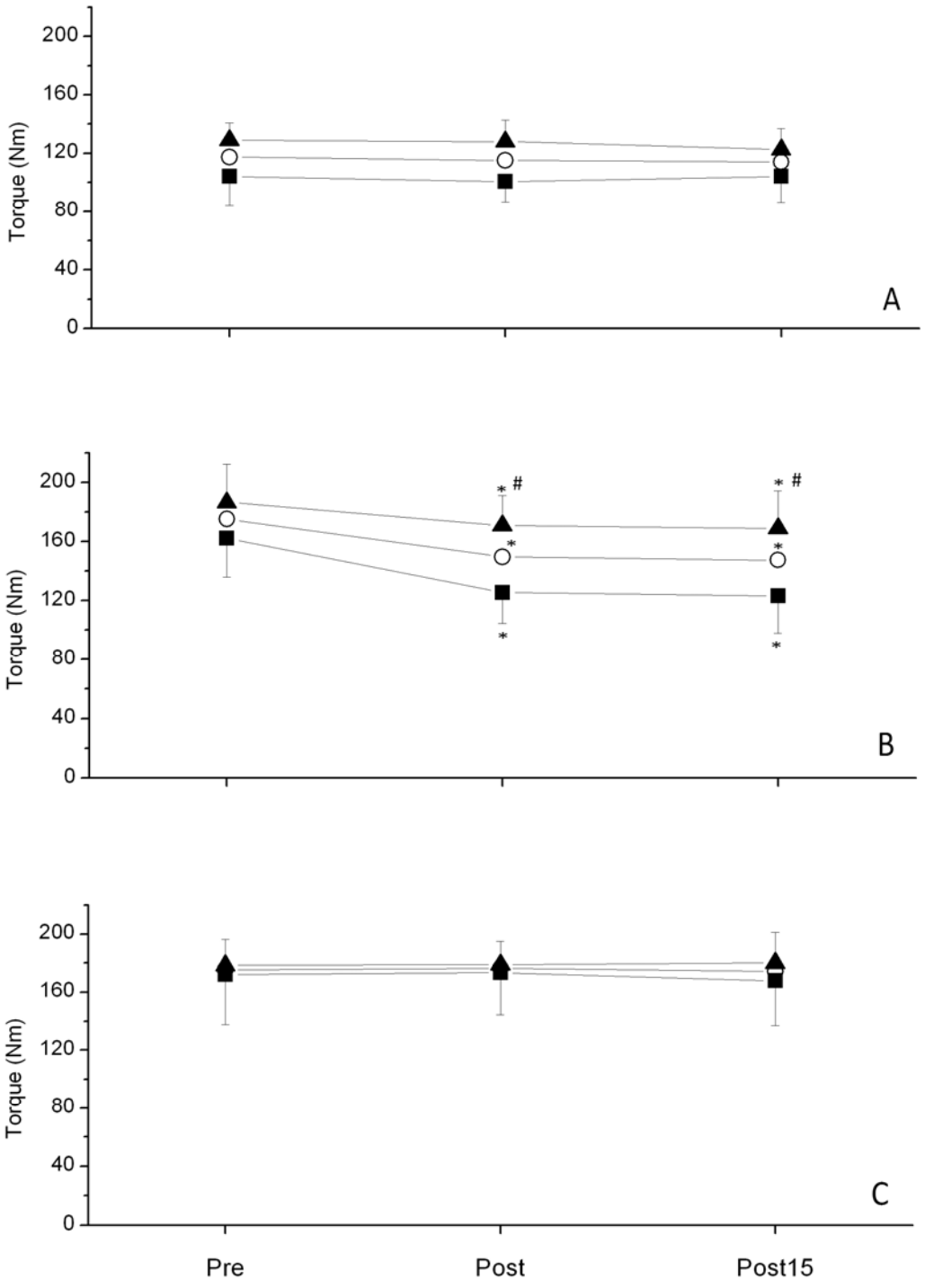

Figure 3-1. Mean concentric knee flexor torque (A), eccentric knee flexor torque (B) and concentric knee extensor torque (C) for low responders (▲), high responders (■) and the whole group (○) before, immediately (Post) and 15 minutes (Post15) after 45 minutes of treadmill running. All torque measurements derived from isokinetic dynamometry at $180^{o}.s^{-1}$. Error bars, not shown for the sake of clarity for whole group data, are standard deviations. * Significantly different from pre-exercise values; # Significant differences between high and low responders; $p < 0.05$.

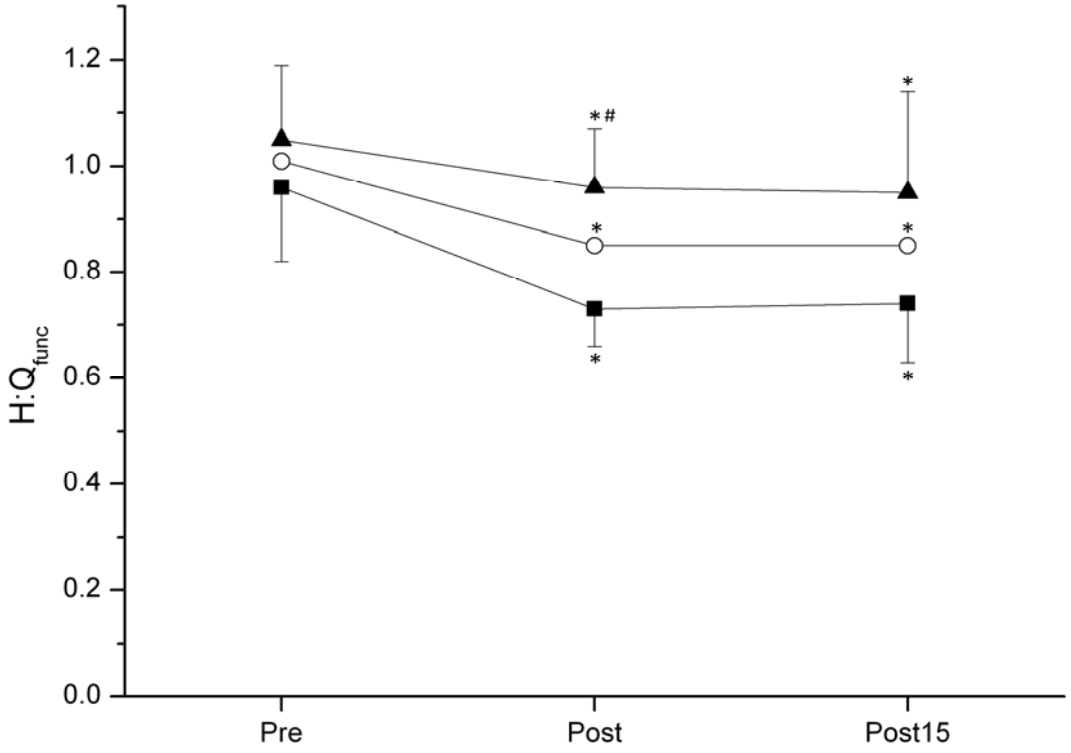

Figure 3-2. Functional hamstrings to quadriceps ratio ($H:Q_{func}$) for low responders (▲), high responders (■) and the whole group (○) before, immediately (Post) and 15 minutes (Post15) after 45 minutes of treadmill running. Error bars, not shown for the sake of clarity for whole group data, are standard deviations. * Significantly different from pre-exercise values; # Significant differences between high and low responders; $p < 0.05$.

3.6 DISCUSSION

Intermittent and repeat sprint running is known to result in a decline in eccentric knee flexor strength and the $H:Q_{func}$. This suggests that the link between prolonged game time in soccer and rugby union and greater likelihood of hamstring strain injuries(9, 15) might be mediated by a decline in eccentric knee flexor function over time. In the current study we investigated whether a greater propensity for muscle damage was a mechanism for this decline in eccentric knee flexor strength following running. As far as the author is aware, this is the first study to explore the relationship between human knee flexor angle of peak torque, as a marker for the propensity of muscle damage, and the extent of knee flexor weakness after soccer specific intermittent running. Because, in the current study we found two distinctly different patterns of knee flexor eccentric strength loss in response to the intermittent treadmill running protocol, we were able to compare angles of knee flexor angle of peak torque and $H:Q_{func}$ between high and low responder groups. The major novel findings from the current study were that 1) angle of peak knee flexor torque and baseline levels of eccentric knee flexor torque did not differ between high and low responders however 2) baseline concentric knee flexor torque measured at $60^{o}.s^{-1}$ was higher in low responders as was concentric knee flexor torque measured at $180^{o}.s^{-1}$ when collapsed across time.

As reported by Greig(39) after a similar running protocol, the strength losses observed here were limited to eccentric actions of the knee flexors. This decline, which averaged approximately 15%, occurred without a concurrent reduction in knee extension strength and suggests a reduced capacity to decelerate the hip and knee

joints during the terminal swing phase of running which may contribute to an increased risk of hamstring strain injury. Indeed, the time course for change in the $H:Q_{func}$ that has been observed after this running protocol(39) fits reasonably well with the increasing injury rates that have been reported as the first and second halves of soccer(15) and rugby union(9) matches progress.

It has been argued that exercise involving powerful eccentric actions, such as the terminal swing phase of running, should be more damaging for muscles that exhibit an angle of peak torque at relatively short lengths because they work to a greater extent along the descending limbs of their length-tension relationships.(76, 145) Lynn, Talbot and Morgan(107) provided evidence consistent with this hypothesis when they reported that rat vastus intermedius muscles with knee angle of peak torque at relatively long lengths were more resistant to strength loss after maximal eccentric actions than muscles whose angles of peak torque occurred at shorter lengths. Furthermore, concentric resistance training, which shifts the torque-joint angle relationship towards shorter muscle lengths,(148) has been reported to increase the strength loss, muscle soreness and damage that occurs after eccentric exercise.(149, 150) Collectively, these observations are consistent with the hypothesis that variation in the length-tension characteristics of muscle alters the susceptibility to damage induced by eccentric contraction. However, in the current study, the pre-exercise angle of peak torque did not differ significantly between participants who exhibited small or large losses in eccentric knee flexor torque. These observations suggest that factors other than angle of peak torque might influence the decline in eccentric knee flexor strength after intermittent running. It is also feasible that the running protocol employed in the current study did not have a

great enough volume or intensity of eccentric contraction to elicit sufficient damage to the hamstring musculature. Protocols involving greater volumes of maximal speed sprinting repetitions may be better suited to explore the role of angle of peak torque on the decline in eccentric knee flexor function following running and should be considered in future studies.

The pre-exercise performance measures that differed significantly between high and low responders in this study were those relating to concentric hamstring strength. Concentric knee flexor torque measured at $60^{o}.s^{-1}$, was significantly higher in low than high responders prior to exercise. Furthermore, concentric knee flexor torque measured at $180^{o}.s^{-1}$, which did not change after exercise, was significantly higher in low than high responders when collapsed across time. Pre-exercise eccentric knee flexor torque measured at $180^{o}.s^{-1}$, did not differ significantly between high and low responders although the ES for this comparison (Cohen's $d = 0.93$) suggests that the current study was underpowered to detect this difference of approximately 15%. This trend towards lower pre-exercise eccentric knee flexor torque in high responders also manifested itself in a trend towards a lower $H:Q_{func}$. The significant differences in concentric hamstring strength and the trends towards differences in eccentric strength are consistent with the possibility that muscular weakness predisposes the knee flexors to larger losses in eccentric strength consequent to intermittent treadmill running. It may also be the case that given the running protocol used the same absolute speeds for all participants, those with greater levels of baseline strength were also those with the highest maximal sprint speeds and as such the intensity of the protocol was lower for these individuals and resulted in lesser declines in strength as a consequence. It should be noted, however, that greater knee flexor strength,

measured via isokinetic dynamometry, does not predict acceleration and maximal sprint speed ability.(151)

As far as we are aware, this is the first study to identify two distinctly different distributions in eccentric knee flexor strength loss after running. While more work is needed to determine how robust these findings are, the range for the decline in eccentric strength noted here (5.1 to 24.9%) is sufficiently large to warrant further enquiry. Fatigue(59) and incomplete activation(58) both reduce skeletal muscle's capacity for energy absorption during active lengthening and this predisposes towards strain injury, at least in isolated muscle. Individuals who lose the greatest amount of eccentric strength may be particularly prone to injury via these mechanisms. We hypothesise that the sensitivity of predicting hamstring injury via the $H:Q_{func}$ would be enhanced if isokinetic tests were conducted in a state of fatigue. An arbitrary cut-off one standard deviation below our mean $H:Q_{func}$ (0.87), identified three of the current subjects as having 'low' $H:Q_{func}$ values prior to exercise while 10 were at or below that level after running. Thus, if the $H:Q_{func}$ ratio can be considered a valid measure of HSI risk,(32, 33) a significant proportion of our participants could be identified as 'at risk' only when tested in a fatigued state. Future prospective studies are needed to determine whether a fatigued $H:Q_{func}$ ratio can better identify athletes at risk of a HSI than tests performed in the unfatigued state.

It is tempting to speculate that the powerful and repetitive eccentric actions performed by the hamstrings during running are in some way responsible for the decline in eccentric knee flexor strength as observed in the current study. The lack of recovery during passive rest, noted here and previously,(39) is entirely consistent

with the effects of purely eccentric exercise(152) and also suggests that metabolic factors had little or no role in causing hamstring weakness. It is, however, unusual to see a substantial decrement in eccentric strength without a concurrent decline in concentric capacity.(153, 154) Repeated eccentric contractions of the elbow flexors(154) and quadriceps(153) have previously been reported to cause similar declines in concentric and eccentric strength. However, these studies employed only relatively slow eccentric speeds ($60^{o}.s^{-1}$)(153, 154) and may not be entirely relevant to studies like this one that employ faster lengthening actions.

There are some limitations associated with the current study. Firstly, we used angle of peak torque of the knee flexors as a measure of propensity for muscle damage, however a number of other methodologies are available to assess the extent of muscle damage, including visual analogue pain scales, girth measurements and serum levels of creatine kinase.(155) We choose the angle of peak torque method as other methodologies are not specific to the knee flexors and can be influenced by damage to other muscles, consequent to intermittent running, such as the quadriceps. Secondly, the assessment of neural function, via electromyography (EMG), of the hamstring musculature may have been warranted to determine any impact of deficits in hamstring muscle activation on eccentric knee flexor weakness observed post running. However, the additional time required to accurately position and/or replace electromyography electrodes would have prohibited a measure of knee flexor strength immediately post the running protocol. Now that we have confirmed that there is no recovery of eccentric knee flexor strength following 15 minutes of passive recovery, work is underway in our laboratory to assess neuromuscular hamstring function 15 minutes following intermittent and repeat sprint running. From the

current findings it would be expected that neuromuscular function of the hamstrings at this time point would be reflective of function immediately following running.

Further work is needed to establish the mechanisms of knee flexor weakness after intermittent running protocols and to determine why some individuals experience significantly more eccentric weakness than others. As shown here, marked variability in eccentric knee flexor strength loss exists without an apparent relationship with the angle of peak torque. Better understanding of the mechanisms responsible would be expected to assist in the development of specific interventions to minimise the extent of eccentric knee flexor strength loss following intermittent running. Such interventions may help to minimise the incidence of HSI given the known link between this injury type and fatigue.

4 STUDY TWO

Knee flexor strength and bicep femoris EMG activity is lower in previously strained hamstrings.

This chapter comprises the following paper accepted for publication in the Journal of Electromyography and Kinesiology:

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The authors listed have certified that:

1. they meet the criteria for authorship in that they have participated in the conception, execution, or interpretation, of at least that part of the publication in their field of expertise;
2. they take public responsibility for their part of the publication, except for the responsible author who accepts overall responsibility for the publication;
3. there are no other authors of the publication according to these criteria;
4. potential conflicts of interest have been disclosed to (a) granting bodies, (b) the editor or publisher of journals or other publications, and (c) the head of the responsible academic unit, and
5. they agree to the use of the publication in the student's thesis and its publication on the QUT ePrints database consistent with any limitations set by publisher requirements.

In the case of this chapter, the following contributions were made:

Contributor	Statement of contribution
David Opar	Determined experimental design, gained ethical approval, participant recruitment, data collection, data analysis, statistical analysis, wrote and submitted the manuscript, responded to reviewer comments approved final proof. Signature: _____ Date: 13/5/13
Morgan Williams	Determined experimental design, statistical analysis, assisted writing the manuscript, responded to reviewer comments approved final proof.
Ryan Timmins	Participant recruitment, data collection, data analysis.
Nuala Dear	Participant recruitment, data collection, data analysis.
Anthony Shield	Determined experimental design, assisted with ethical approval, assisted writing the manuscript.

Principal Supervisor Confirmation

I have sighted email or other correspondence from all Co-authors confirming their certifying authorship.

ANTHONY SHIELD A. Shield 13/5/13

Name Signature Date

4.1 LINKING PARAGRAPH

The eccentric specific declines in hamstring strength noted in response to intermittent running in Chapter 3 is similar to reports which have shown a previously strained hamstring displays deficits in eccentric strength compared to a contralateral uninjured limb. Much like the declines in eccentric strength following intermittent running the mechanism remains unknown. Chapter 4 focuses on whether deficits in neural function play some role in this prolonged decline in eccentric hamstring strength following hamstring strain injury (HSI).

4.2 OVERVIEW

The aim of this study was to determine if athletes with a history of HSI display lower levels of surface EMG (sEMG) activity and median power frequency in the previously injured hamstring muscle during maximal voluntary contractions. Recreational athletes were recruited, 13 with a history of unilateral HSI and 15 without prior injury. All athletes undertook isokinetic dynamometry testing of the knee flexors and sEMG assessment of the biceps femoris long head (BF_L) and medial hamstrings (MH) during concentric and eccentric contractions at ± 180 and ± $60^o.s^{-1}$. The knee flexors on the previously injured limb were weaker at all contraction speeds compared to the uninjured limb (+$180^o.s^{-1}$, p = 0.0036, ES = 0.78; +$60^o.s^{-1}$, p = 0.0013, ES = 0.70; -$60^o.s^{-1}$, p = 0.0007, ES = 0.57; -$180^o.s^{-1}$, p = 0.0007, ES = 0.74) whilst sEMG activity was only lower in the BF_L during eccentric contractions (-$60^o.s^{-1}$, p = 0.0025, ES = 0.47; -$180^o.s^{-1}$, p = 0.0003, ES = 0.58). There were no between limb differences in MH sEMG activity or median power frequency from either BF_L or MH in the injured group. The uninjured group showed no between limb differences in any of the tested variables. Secondary analysis comparing the between limb difference in the injured and the uninjured groups, confirmed that previously injured hamstrings were mostly weaker (+$180^o.s^{-1}$, p = 0.2208, ES = 0.48; +$60^o.s^{-1}$, p = 0.0379, ES = 0.83; -$60^o.s^{-1}$, p = 0.0312, ES = 0.85; -$180^o.s^{-1}$, p = 0.0110, ES = 1.03) and that deficits in sEMG were confined to the BF_L during eccentric contractions (-$60^o.s^{-1}$, p = 0.0542, ES = 0.74; -$180^o.s^{-1}$, p = 0.0473, ES = 0.82) Previously injured hamstrings were weaker and BF_L sEMG activity was lower than the contralateral uninjured hamstring. This has implications for HSI prevention and rehabilitation which should consider altered neural function following HSI.

4.3 INTRODUCTION

HSIs, characterised by acute pain in the posterior thigh and disruption of hamstring muscle fibres, are the primary injury sustained in a number of sports(7, 15, 19) and re-injury rates are also high.(7) The high rate of injury and re-injury, combined with the fact that a previous HSI is the most significant risk factor for future injury,(42) suggests that our understanding of the neuromuscular maladaptations that occur following HSI requires further attention.

Previous HSI has been associated with between-limb differences in eccentric strength that is typically greater than concentric strength deficits.(43, 45) Furthermore, these deficits in eccentric strength are still present despite athletes returning to full training and competition.(43, 45) Whilst the retrospective nature of these findings cannot be taken to suggest that HSI has resulted in these deficits, it is agreed that HSI does lead to maladaptation.(142) Importantly, prospective studies in both sprinters and soccer players have identified eccentric knee flexor strength deficits as elevating HSI risk.(32, 33) These findings suggest the importance of eccentric strength for the prevention of HSI and that eccentric weakness should be corrected following injury to reduce the risk of a recurrence. However, a clear understanding of the mechanisms underpinning the decline in eccentric strength following HSI is required in order to develop more appropriate exercise interventions. Whilst evidence does exist of persistent atrophy of biceps femoris long head (BF_L) up to 23 months following grade I and II HSI (46) this muscular maladaptation does not explain why the decline in hamstring strength appears to be greater in eccentric actions.(43, 45)

Surprisingly, the impact of strain injuries on the neural function of the involved musculature has been largely overlooked. HSI has been reported to result in acute(20) and chronic pain.(43, 44) This muscular pain also has the potential to alter central nervous function at both the spinal and supraspinal level,(156) and might therefore be expected to result in a restriction of EMG activity and the median power frequency of this activity during contraction. Furthermore, this restriction may be specifically confined to the muscle and contraction mode responsible for the noxious stimulus. Therefore the purpose of this study was to assess concentric and eccentric hamstring torque, sEMG activity and the median power frequency of the sEMG signal of recreational athletes with and without a history of unilateral HSI. It was hypothesised that the previously injured hamstrings would display strength, sEMG activity and median power frequency deficits during fast and slow eccentric contractions, but not concentric contractions, compared to the contralateral limb. Furthermore, we hypothesised that lower levels of sEMG activity and median power frequency would be confined specifically to the previously injured hamstring muscle (i.e. BF_L or MH). It was also hypothesised that the control group would display no differences in any of the aforementioned variables between dominant and non-dominant limbs. As a confirmatory secondary analysis, it was also hypothesised that the between limb differences in eccentric hamstring torque, sEMG and median power frequency would be greater in previously injured athletes compared to the control group.

4.4 METHODS

4.4.1 PARTICIPANTS

Twenty-eight recreationally active males participated in the study, with most competing in Australian football, rugby, soccer or sprinting. Thirteen athletes (26.2 ± 5.8 years; 1.80 ± 0.04m; 83.0 ± 14.8kg) had at least one unilateral HSI within the last 18 months and all had suffered a grade II injury previously. Another 15 athletes (26.7 ± 5.8 years; 1.8 ± 0.05m; 83.5 ± 7.9 kg) had no history of HSI. All participants were free of any other injury to the lower limbs and were fully active in their chosen sport at the time of testing. All testing procedures were approved by the University Human Research Ethics Committee. Participants gave informed written consent prior to testing after having all procedures explained to them.

4.4.2 PARTICIPANT INJURY HISTORIES

Following recruitment, participants completed an injury questionnaire with their chosen practitioner (i.e. physiotherapist) who had previously diagnosed and treated all the athletes HSI. As per previous investigations,(130) the notes taken from clinical examination were used to detail the date of injury and return to pre-injured levels of training and competition, severity (grade I, II or III),(21) location (dominant or non-dominant limb; BF_L or MH head; proximal or distal) and rehabilitation details of all previous HSIs. Limb dominance was determined as the preferred kicking limb. Athletes were considered to be successfully rehabilitated when they returned to pre-injured levels of training and were available for competition.(157) Athletes who were unable to obtain data on all prior hamstring strains from their practitioner were excluded from the study.

4.4.3 sEMG

Bipolar pre-gelled silver/silver-chloride sEMG electrodes (10mm diameter, 25mm inter-electrode distance) were used to record EMG activity from the MH and BF_L. After preparation of the skin via shaving, light abrasion and sterilisation, electrodes were placed on the posterior thigh half way between the ischial tuberosity and tibial epicondyles with electrodes oriented parallel to the line between these two land marks, as per Surface Electromyography for the Non-Invasive Assessment of Muscles guidelines.(158) The reference electrode was placed on the ipsilateral head of the fibula. Muscle bellies were identified via palpation during forceful isometric knee flexion and correct placement was confirmed by observing sEMG activity during active internal and external rotation of the flexed knee to assess cross talk between MH and BF_L.

4.4.4 ISOKINETIC DYNAMOMETRY

Assessment of concentric and eccentric knee flexor strength was performed on a Biodex Systems 3 Dynamometer (Biodex Medical Systems, Shirley, NY). Participants were seated on a custom pad, placed on top of the original seat, which contained two holes at the level of the posterior mid thigh to minimise movement artefact from sEMG electrodes on the dynamometer seat. The hips were flexed at 85^o from neutral with the lateral epicondyle of the femur carefully aligned with the fulcrum of the dynamometer. The tested leg was attached to the lever of the dynamometer via a Velcro strap and padded restraints were fastened across the trunk, hips and mid thigh of the tested leg to isolate movement to the knee joint. The range of motion was set at 5^o-90^o of knee flexion (0^o=full knee extension) and correction

for limb weight was performed. Three sets of four submaximal contractions of the knee extensors and flexors were performed at $+240^{o}.s^{-1}$ as a warm-up to prepare the participant for maximal effort in the following sets. Concentric testing for both legs consisted of three sets of three consecutive maximum voluntary contractions of the knee extensors and flexors at speeds of $+60^{o}.s^{-1}$ and $+180^{o}.s^{-1}$ with 30 seconds rest between sets. Athletes were motivated verbally by the investigators to encourage maximal effort throughout the range of motion. Eccentric testing ($-60^{o}.s^{-1}$ and $-180^{o}.s^{-1}$) was identical except that only eccentric contraction of the knee flexors was performed by the participant (whereby the knee joint was extended despite active contraction of the knee flexors) and at the completion of each contraction the investigators returned the lever to the starting position. The leg and speed testing orders were randomised but concentric contractions were always performed before eccentric contractions. All participants were required to attend at least one familiarisation session to ensure consistency of maximum voluntary contractions and one testing session with ≥ seven days between sessions.

4.4.5 DATA ANALYSIS

Dynamometer torque and lever position data were transferred to computer at 1 kHz and stored for later analysis. Average peak torque was defined as the mean maximal torque of the six highest torque contractions at each velocity. sEMG was sampled simultaneously with dynamometer data at 1kHz through a 16-bit PowerLab26T AD recording unit (ADInstruments, New South Wales, Australia) (amplification = 1000 between 10Hz-1kHz; common mode rejection ratio = 110dB) and stored for later analysis where it was fourth order Butterworth filtered between 20-500Hz (24dB roll

off) using MATLAB (MathWorks, Natick, Massachusetts) and then full wave rectified using the root-mean-square method and smoothed using a 100-point moving average. At each velocity, sEMG data were averaged across a knee joint range of motion between 15^{o}-35^{o} as this is where deficits in sEMG have been noted previously.(130) Data at all velocities was then normalised to the maximal averaged sEMG amplitude recorded during MVCs at $+180^{o}.s^{-1}$.(159-161) For this process the data was separated in tertiles throughout the ROM (15^{o}-35^{o}, 35^{o}-60^{o}, 60^{o}-80^{o}) and the tertile exhibiting the highest amplitude of sEMG was used for normalisation. Median power frequency was determined from the non-rectified sEMG signal via Fast Fourier transform with Hann window function applied(136) across the entire ROM using LabCart 7.3 (ADInstruments, New South Wales, Australia) with 1Hz frequency resolution. This resulted in 1.08 and 0.36 second time epochs for analysis of contractions at ± 60^{o} and $180^{o}.s^{-1}$ respectively. Median power frequency was analysed over a larger ROM (15-80^{o}) than sEMG activity to allow for a valid estimation of frequency. Median power frequency was defined as the frequency at which 50% of total power was reached for each time epoch.

4.4.6 STATISTICAL ANALYSIS

Data were analysed using JMP version 10.0 Pro Statistical Discovery Software (SAS Inc). In the primary analysis, comparisons were made between the injured and uninjured limbs in the injured group and between dominant and non-dominant limbs in the uninjured group. Dependent variables were compared using one tailed paired t tests for both groups to allow an equal likelihood for finding significant differences between limbs.(45) Data are presented as means and standard deviation. Bonferroni

corrections were performed to account for four comparisons made for each dependent variable across the velocities used, with significance set at $p < 0.0125$. In the confirmatory secondary analysis independent t tests for unequal variance were used to compare the between limb differences of the dependent variables in the injured (uninjured limb minus injured limb) and uninjured group (dominant limb minus non-dominant limb) as assumptions for equal variance between groups was not met. For the secondary analysis significance was set at $p < 0.05$ and data are presented as mean differences and 95% confidence intervals (95% CI). To assess the magnitudes of the differences for the primary and secondary analyses Cohen's d was calculated to report effect size (ES).

4.5 RESULTS

4.5.1 PARTICIPANTS

There was no significant difference between the injured and uninjured groups with respect to age, height or body mass. The details of injury histories of all athletes from the injured group can be found in Table 4-1. All athletes from the injured group reported largely standard rehabilitation progression (i.e. Ref (131)) guided by their physiotherapist.

4.5.2 KNEE FLEXOR STRENGTH

There were significant differences in average peak torque between limbs in the injured group, with the previously injured limb weaker at all contraction modes and velocities (Figure 4-1a & Table 4-2). No differences in average peak torque were noted between limbs in the uninjured group (Figure 4-1b & Table 4-2). Between limb differences in torque were significantly greater in the injured group compared to the uninjured group at all contraction modes and velocities, except for concentric contractions at $180^{o}.s^{-1}$ (Table 4-7).

4.5.3 HAMSTRING sEMG ACTIVITY

BF_L sEMG activity was significantly lower in the previously injured limb compared to the contralateral uninjured limb in the injured group during eccentric contractions but not concentric contractions (Figure 4-2a & Table 4-3). There were no differences between limbs in the injured group for MH sEMG activity at any contraction mode or velocity (Figure 4-3a & Table 4-3). In the uninjured group there were no differences in sEMG between limbs for BF_L (Figure 4-2b & Table 4-4) or MH

(Figure 4-3b & Table 4-4) at any contraction mode or velocity. Between limb differences in sEMG activity were greater in the injured group compared to the uninjured group only for BF_L at $-180^o.s^{-1}$ (Table 4-8). All other between limb differences in sEMG activity were similar between injured and uninjured groups, although a trend existed in BF_L at $-60^o.s^{-1}$ (Table 4-8).

4.5.4 MEDIAN POWER FREQUENCY OF HAMSTRING sEMG

One participant from the injured group was a clear outlier (median power frequency was more than 3 standard deviations above the mean for eccentric contractions) and was removed from analysis. There were no differences in median power frequency at any velocity between legs in the injured group for BF_L or MH (Table 4-5). A similar lack of differences was noted at all velocities for the uninjured group for BF_L or MH median power frequency (Table 4-6). The between limb differences in median power frequency did not differ between the injured and uninjured groups at any contraction mode or velocity.

Table 4-1. Hamstring strain injury information for most recent injury for athletes recruited to the injured group.

Participant	Time since HSI (months)	Rehabilitation duration (weeks)	Location	Total HSIs sustained
1	2	4	Dom, Prox BF_L	1
2	3	4	Non-dom, Prox BF_L	3
3	8	4	Non-dom, Dist BF_L	1
4	7	2	Non-dom, Prox BF_L	2
5	3	4	Dom, Prox BF_L	4
6	5	2	Non-dom, Dist BF_L	2
7	18	4	Non-dom, Dist BF_L	1
8	4	4	Non-dom, Prox BF_L	2
9	2	5	Non-dom, Prox BF_L	2
10	5	3	Non-dom, Prox BF_L	4
11	2	2	Dom, Prox BF_L	2
12	3	6	Non-dom, Dist BF_L	4
13	7	3	Non-dom, Prox BF_L	3

HSI, hamstring strain injury; Dom, dominant limb; Non-dom, non dominant limb; Prox, proximal; Dist, distal; BF_L, biceps femoris. All prior injuries were confined to the same leg and muscle as most recent injury however location on muscle (proximal or distal) differed in some instances.

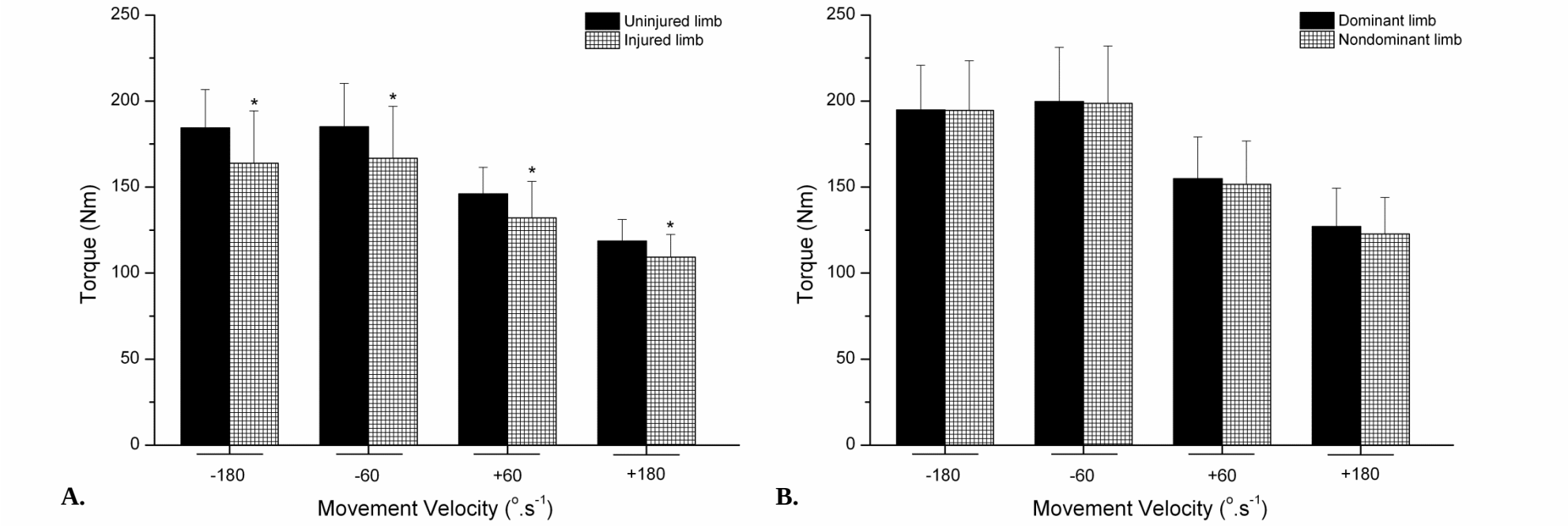


Figure 4-1. Knee flexor average peak torque at four different isokinetic velocities from the A) injured athletes and B) uninjured athletes. Negative movement velocities are indicative of eccentric contractions and positive velocities indicate concentric contractions. Error bars display standard deviation. * $p < 0.0125$ injured vs. uninjured limbs.

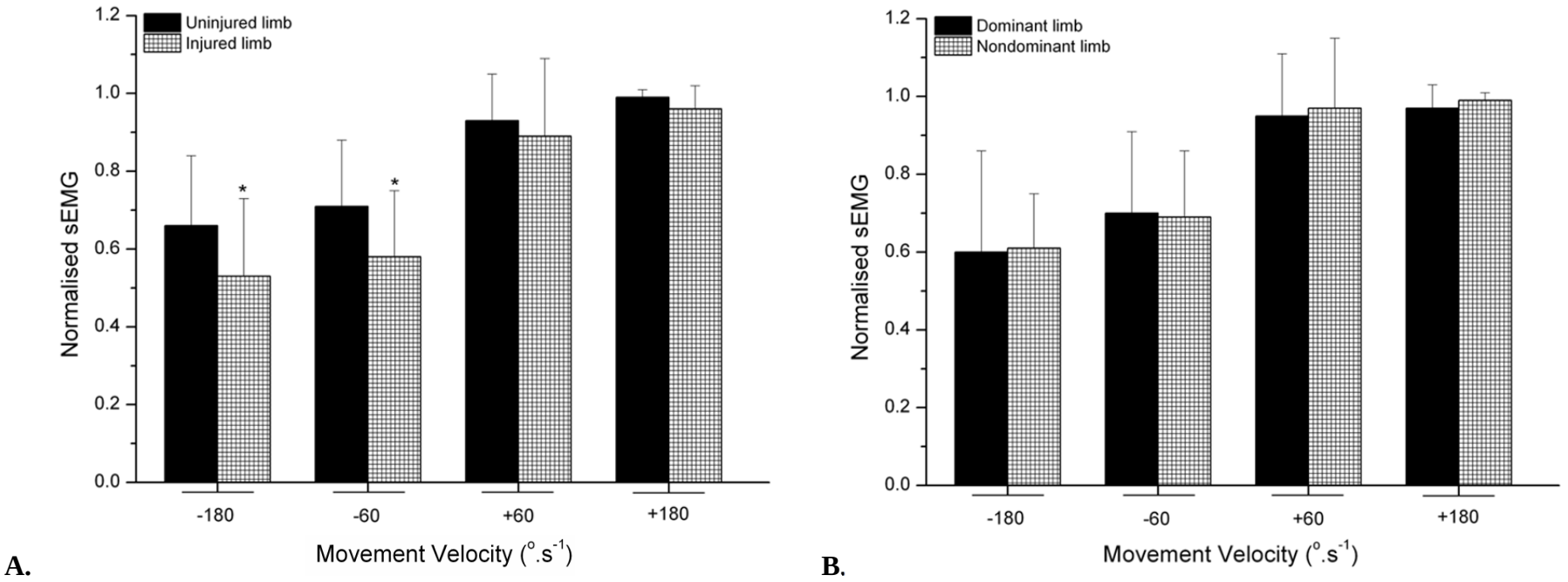


Figure 4-2. Biceps femoris long head normalised surface electromyography (sEMG) at four different isokinetic velocities from the A) injured athletes and B) uninjured athletes. Negative movement velocities are indicative of eccentric contractions and positive velocities indicate concentric contractions. Error bars display standard deviation.* $p < 0.0125$ injured vs. uninjured limbs.

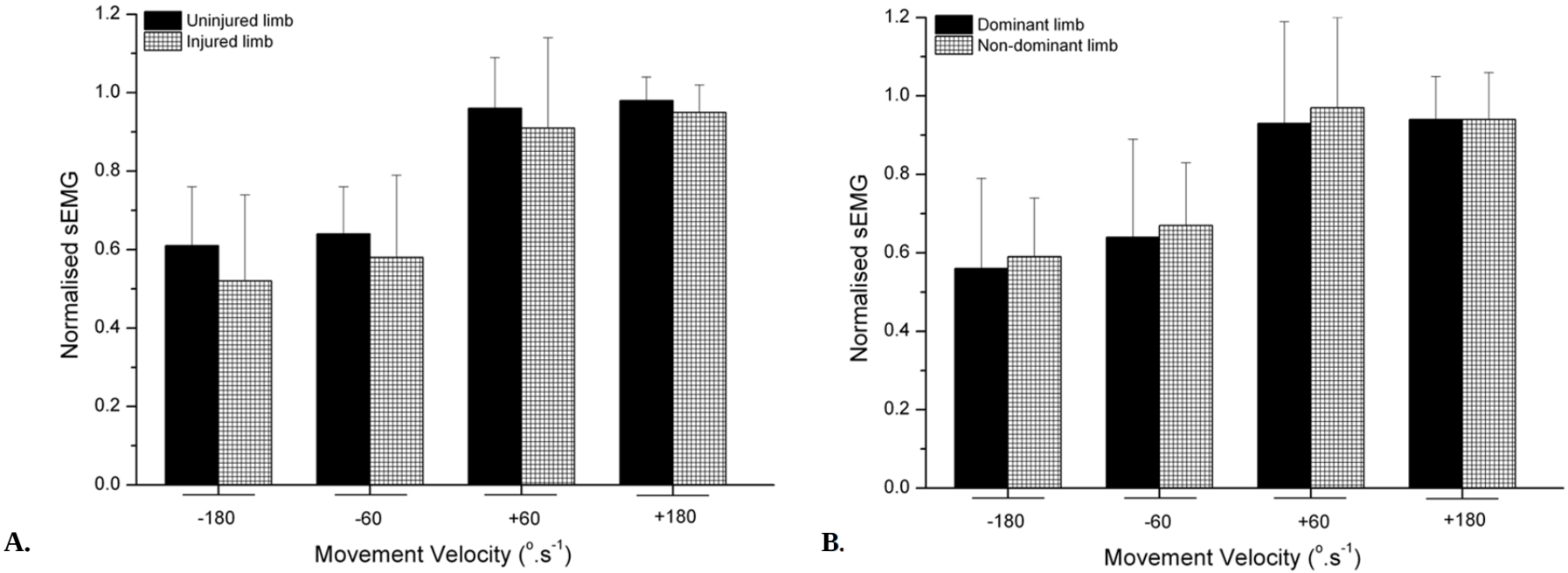


Figure 4-3. Medial hamstring normalised surface electromyography (sEMG) at four different isokinetic velocities from the A) injured athletes and B) uninjured athletes. Negative movement velocities are indicative of eccentric contractions and positive velocities indicate concentric contractions. Error bars display standard deviation.

Table 4-2. Knee flexor torque of athletes with and without a history of unilateral hamstring strain injury during concentric and eccentric contraction.

Movement velocity	Injured Group			
	Injured limb	Uninjured limb	p	ES
+180	109.29 (± 13.14)	118.64 (± 12.47)	0.0036*	0.78
+60	132.00 (± 21.28)	146.01 (± 15.49)	0.0013*	0.70
-60	166.76 (± 30.19)	185.02 (± 25.22)	0.0007*	0.57
-180	163.82 (± 30.43)	184.37 (± 22.33)	0.0007*	0.74
Movement velocity	Uninjured group			
	Dominant limb	Non-dominant limb	p	ES
+180	127.13 (± 22.12)	122.73 (± 21.24)	0.0608	0.20
+60	154.93 (± 24.27)	151.59 (± 25.10)	0.1558	0.14
-60	199.71 (± 31.46)	198.68 (± 33.30)	0.4341	0.03
-180	194.84 (± 25.97)	194.60 (± 28.84)	0.4828	0.01

Negative movement velocities are indicative of eccentric contractions and positive velocities indicate concentric contractions. Data are presented as mean (± standard deviation). *Significance was set at $p < 0.0125$. Cohen's d was used to calculate effect size (ES).

Table 4-3. The normalised electromyography of the biceps femoris long head and medial hamstrings of athletes with a history of hamstring strain injury.

Movement velocity	Injured group							
	Biceps femoris long head				Medial hamstrings			
	Injured limb	Uninjured	p	ES	Injured limb	Uninjured	p	ES
+180	0.96 (±0.06)	0.99 (± 0.02)	0.0894	[a]	0.95 (± 0.07)	0.98 (± 0.06)	0.0622	[a]
+60	0.89 (± 0.20)	0.93 (± 0.12)	0.2255	0.18	0.91 (± 0.23)	0.96 (± 0.13)	0.2412	0.09
-60	0.58 (± 0.17)	0.71 (± 0.17)	0.0025*	0.47	0.58 (± 0.21)	0.64 (± 0.12)	0.1296	0.06
-180	0.53 (± 0.20)	0.66 (± 0.18)	0.0003*	0.58	0.52 (± 0.22)	0.61 (± 0.15)	0.0770	0.26

Negative movement velocities are indicative of eccentric contractions and positive velocities indicate concentric contractions. Data are presented as mean (± standard deviation). *Significance was set at $p < 0.0125$. Cohen's d was used to calculate effect size (ES). [a] ES for electromyographical activity could not be calculated given the use of this data in the normalisation process.

Table 4-4. The normalised electromyography activity of the biceps femoris long head and medial hamstrings of athletes without a history of hamstring strain injury.

Movement velocity	Uninjured group							
	Biceps femoris long head				Medial hamstrings			
	Dominant	Non-	P	ES	Dominant	Non-	P	ES
+180	0.97 (±0.06)	0.99 (± 0.02)	0.1602	[a]	0.95 (± 0.07)	0.98 (± 0.06)	0.0622	[a]
+60	0.95 (± 0.16)	0.97 (± 0.18)	0.2703	0.18	0.91 (± 0.23)	0.96 (± 0.13)	0.2412	0.09
-60	0.58 (± 0.17)	0.71 (± 0.17)	0.0025*	0.47	0.58 (± 0.21)	0.64 (± 0.12)	0.1296	0.06
-180	0.53 (± 0.20)	0.66 (± 0.18)	0.0003*	0.58	0.52 (± 0.22)	0.61 (± 0.15)	0.0770	0.26

Negative movement velocities are indicative of eccentric contractions and positive velocities indicate concentric contractions. Data are presented as mean (± standard deviation). Cohen's d was used to calculate effect size (ES). [a] ES for electromyographical activity could not be calculated given the use of this data in the normalisation process.

Table 4-5. Median power frequency of the biceps femoris long head and medial hamstrings of athletes with a history of unilateral hamstring strain injury during concentric and eccentric contraction.

Movement velocity	Injured group							
	Biceps femoris long head				Medial hamstrings			
	Injured limb	Uninjured limb	P	ES	Injured limb	Uninjured limb	P	ES
+180	61.70 (± 5.82)	64.70 (± 9.00)	0.1005	0.40	67.75 (± 6.25)	71.15 (± 8.34)	0.1680	0.47
+60	60.30 (± 6.64)	62.11 (± 7.80)	0.2220	0.25	58.70 (± 7.48)	62.78 (± 9.57)	0.1655	0.48
-60	64.78 (± 7.83)	66.92 (± 9.35)	0.2530	0.24	62.85 (± 9.63)	66.03 (± 15.53)	0.2950	0.25
-180	63.04 (± 6.38)	68.03 (± 13.73)	0.1030	0.50	64.68 (± 9.42)	70.43 (± 18.49)	0.2140	0.41

Negative movement velocities are indicative of eccentric contractions and positive velocities indicate concentric contractions. Data are presented as mean standard deviation). Significance was set at $p < 0.0125$. Cohen's d was used to calculate effect size (ES).

Table 4-6. Median power frequency of the biceps femoris long head and medial hamstrings of athletes without a history of unilateral hamstring strain injury during concentric and eccentric contraction.

Movement velocity	Uninjured group							
	Biceps femoris long head				Medial hamstrings			
	Dominant limb	Non-dominant limb	P	ES	Dominant limb	Non-dominant	P	ES
+180	63.57 (± 11.35)	62.82 (± 7.41)	0.3580	0.08	74.84 (± 13.24)	72.04 (± 7.71)	0.2460	0.26
+60	62.71 (± 7.60)	62.84 (± 7.51)	0.4670	-0.02	69.44 (± 10.44)	66.28 (± 6.28)	0.1025	0.37
-60	63.25 (± 9.37)	63.38 (± 6.89)	0.4620	-0.02	70.24 (± 15.52)	66.42 (± 13.50)	0.2075	0.26
-180	64.22 (± 12.62)	66.05 (± 8.26)	0.2400	-0.17	70.21 (± 18.21)	71.05 (± 13.62)	0.4275	-0.05

Negative movement velocities are indicative of eccentric contractions and positive velocities indicate concentric contractions. Data are presented as mean (± standard deviation). Significance was set at $p < 0.0125$. Cohen's d was used to calculate effect size (ES).

Table 4-7. Comparison of between limb differences in knee flexor torque in athletes with and without a history of hamstring strain injury, during concentric and eccentric contraction.

Movement velocity	Knee flexor torque			
	Injured group	Uninjured group	P	ES
+180	9.34 (3.03 to 15.66)	4.40 (-1.33 to 10.13)	0.2208	0.48
+60	14.01 (5.98 to 22.02)	3.34 (-3.48 to 10.16)	0.0379*	0.83
-60	18.26 (8.68 to 27.84)	1.03 (-12.10 to 14.17)	0.0312*	0.85
-180	20.55 (9.72 to 31.37)	0.24 (-11.56 to 12.04)	0.0110*	1.03

Negative movement velocities are indicative of eccentric contractions and positive velocities indicate concentric contractions. Data are presented as mean differences (95% confidence intervals). *Significance was set at $p < 0.05$. Cohen's d was used to calculate effect size (ES).

Table 4-8. Comparison of between limb differences in normalised electromyographical activity of the biceps femoris long head and medial hamstrings in athletes with and without a history of hamstring strain injury, during concentric and eccentric contraction.

Movement velocity	Normalised electromyographical activity							
	Biceps femoris long head				Medial hamstrings			
	Injured group	Uninjured group	P	ES	Injured group	Uninjured group	P	ES
+180	0.03 (-0.01 to 0.07)	-0.01 (-0.05 to 0.02)	0.0919	[a]	0.03 (-0.01 to 0.06)	0.00 (-0.08 to 0.07)	0.4070	[a]
+60	0.04 (-0.07 to 0.15)	-0.03 (-0.11 to 0.06)	0.3271	0.41	0.05 (-0.10 to 0.21)	-0.04 (-0.17 to0.10)	0.3661	0.36
-60	0.13 (0.05 to 0.22)	0.01 (-0.09 to 0.11)	0.0542	0.74	0.07 (-0.06 to 0.20)	-0.03 (-0.15 to 0.09)	0.2395	0.46
-180	0.13 (0.07 to 0.19)	-0.02 (-0.15 to 0.12)	0.0473*	0.82	0.09 (-0.04 to 0.21)	-0.03 (-0.13 to 0.07)	0.1210	0.61

Negative movement velocities are indicative of eccentric contractions and positive velocities indicate concentric contractions. Data are presented as mean differences (95% confidence intervals). *Significance was set at $p < 0.05$. Cohen's d was used to calculate effect size (ES). [a] ES for electromyographical activity could not be calculated given the use of this data in the normalisation process.

4.6 DISCUSSION

It is accepted that a prior HSI results in maladaptation of the previously injured tissue.(142) Whilst a number of muscular maladaptations have been reported previously,(43, 45, 46, 76, 95, 101) the impact of a prior HSI on neural function has been scarcely examined.(130) The current study used between limb comparisons of normalised sEMG activity and median power frequency to determine differences in neural hamstring function between injured and uninjured limbs. This method eliminates a number of confounding factors by ensuring that muscle lengths and electrode locations are identical between trials within and between limbs and has been used extensively to assess relative muscle activation in maximal concentric and eccentric contraction.(159-161)

From the injured group in the current study, the novel findings were that the previously injured limb, when compared to the contralateral uninjured limb displayed 1) a lower level of sEMG activity specifically in the previously injured muscle (BF_L) during slow and fast eccentric contractions; and; 2) there was no difference in the median power frequency in either the previously injured BF_L or uninjured MH. Furthermore, lower levels of strength were observed across all contraction modes and velocities in the injured limb compared to the uninjured limb in the injured group. In contrast, the uninjured group showed no differences between dominant and non-dominant limbs in any of the tested variables indicating there is no influence of limb dominance. These findings were mostly supported by confirmatory analysis which indicated that the between limb differences in knee flexor torque at all contraction modes and velocities, except for the fastest concentric contractions, and

BF_L sEMG during fast eccentric contraction was greater in injured group compared to the uninjured group.

This study is, to our knowledge, the first to identify lower levels of sEMG activity specifically in the previously injured BF_L muscle compared to a contralateral uninjured BF_L. Recent evidence examining a similar phenomenon did not find a muscle specific, between limb differences in sEMG activity following a HSI.(130) The discrepancies between the findings from the current study and the previous study by Sole and colleagues(130) work may be attributed to the inclusion of athletes with bilateral injury histories which may have contributed to the lack of difference in sEMG activity between the injured leg and the contralateral control limb in earlier work.(130) However our finding that, when comparing BF_L sEMG across the two groups, only during eccentric contractions at $-180^{o}.s^{-1}$ was the between limb difference significantly greater in the injured compared to the uninjured group, somewhat confirms a previous similar finding by Sole *et al.*(130) Whilst there was no significant between limb difference in BF_L sEMG during eccentric contractions at $-60^{o}.s^{-1}$ when comparing the two groups in the current study, the large ES (Cohen's *d* = 0.74) indicates that a significant difference may have existed with an increased sample size.

Reductions in muscle activation during eccentric contractions is due to reduced motor unit recruitment and/or firing rates(162) which impact upon maximal torque generation capabilities. Following HSI it has been suggested that the purpose of reduced hamstring activation would be to protect the damaged tissue from high force

contraction.(142) HSIs themselves are characterised by acute pain in the posterior thigh(20) with reports of chronic pain not uncommon(43, 44) and this has the potential to result in long-term re-organisation of the nervous system at the spinal and supraspinal levels.(156) The current study confirms that, even in athletes who have been successfully rehabilitated and have returned to competition, sEMG activity of the BF_L remains suppressed. This would indicate that, for the current cohort, contemporary rehabilitation practices were unsuccessful at addressing deficits in the activation of BF_L. This is of concern from the perspective of HSI recurrence given submaximal stimulation of *in-situ* animal muscle reduces the amount of stress that muscle can withstand before the occurrence of stretch induced failure.(58) This may indicate that the previously injured BF_L is unable to withstand the same amount of stress before failure compared to an uninjured muscle, thus increasing the likelihood of re-injury. The observation of no between limb differences in median power frequency in the injured group suggests that prior HSI may not impact upon average muscle fibre conduction velocity.(163) It should also be acknowledged that a number of other factors also influence the median power frequency of the EMG signal and further investigation examining these factors discretely is warranted.

It has been proposed previously that the suppression of hamstring muscle activation following strain injury has the potential to limit adaptation during the rehabilitation process.(142) This model suggests early to middle stage rehabilitation for HSI typically involves avoidance of excessive stretching of the involved tissue and submaximal exercise performed through limited range of motion in an attempt to prevent proliferation of scar tissue.(131) Such an approach might be expected to

result in a reduction of in-series sarcomeres(132) and induce atrophy(46) potentially reducing the optimal length of the hamstrings(76) which would be unfavourable given the need for the hamstrings to generate high eccentric forces at relatively long muscle lengths in running.(37) Late stage rehabilitation involving more forceful eccentric contractions at long muscle lengths might be expected to overcome these maladaptations,(133) however, suppression of hamstring activation, as reported in the current study, would reduce the stimulus the previously injured muscle is exposed to, thus potentially compromising the adaptive response to rehabilitation. The present study suggests that chronic lowering of hamstring activation following strain injury could sabotage the rehabilitation process. Still, the full impact of prior hamstring strain injury on neurological control of the involved muscle/s and impact on adaptation requires further attention.

The current study found strength at all speeds and contraction modes was lower in the previously injured limb compared to the uninjured limb. Previous work has found eccentric but not concentric declines in strength(45) or greater eccentric deficits (22-24%) compared to concentric deficits (10-11%) following HSI.(43) As muscle shortening velocity is known to influence maximal tension generating capacity(164) the different concentric velocities used in previous work may explain the inconsistent findings for this contraction mode. In line with this, the percentage difference in strength between previously injured and uninjured limbs tested at a comparable speed ($+60^{o}.s^{-1}$) is similar in the current study (10.9%) and previous work (11%).(43) The much larger decline in eccentric strength reported elsewhere(43) is less likely to be due to differences in eccentric testing speed as eccentric strength is largely

unaffected by lengthening velocity. It may be, however, explained by differences in rehabilitation practices of the respective cohorts given the greater appreciation for eccentric conditioning in HSI prevention in recent times.(143) Perhaps not surprisingly, more recent studies have reported smaller eccentric strength differences in the order of 13%,(45) which is comparable to the 10.9-12.5% differences reported in the current study.

Uniformly lower concentric and eccentric strength, as observed in the current study, would be expected if strength was determined solely from muscle cross sectional area and volume, given the noted atrophy of BF_L following HSI.(46) Interestingly, sEMG activity was lower only during eccentric contractions, despite lower strength across all contraction modes and velocities. This suggests that reductions in BF_L activity contribute to prolonged eccentric, but not concentric, weakness following HSI. It might therefore be expected that the decline in eccentric strength following HSI would be of a greater relative magnitude than concentric strength, but this is not supported by the current data. It may be that other muscles which contribute to knee flexion, that were not examined in the current study, such as the short head of biceps femoris (BF_S), gastrocnemius and sartorius, increase their involvement during maximal eccentric contraction in a previously injured leg to help overcome the limitation in sEMG activity of BF_L. Indeed, compensatory hypertrophy of BF_S has been reported previously,(46) suggesting HSI may lead to increased use of uninjured musculature, however further examination of this area is warranted.

There are some limitations in the present study's methodology. The retrospective nature of the study does not allow for the determination of whether the reduction in sEMG activity of BF_L is the cause of or the result of injury. Prospective studies are required to determine if low levels of BF_L activity elevates the risk of sustaining a future HSI. It should be noted, however, that whilst prospective studies have determined that a between limb eccentric strength difference of approximately 4.5% is associated with future HSI,(33) post-injury eccentric weakness is reported to be between 13-24%,(43, 45) suggesting HSI enhances eccentric knee flexor weakness, most probably via neuromuscular maladaptation. Also using the maximal activation data from the fastest concentric movement speed ($+180^{o}.s^{-1}$) to normalise the sEMG data as per previous investigations(136) has the potential to mask any between-limb differences in sEMG activity at this speed, however given the important nature of eccentric strength in HSI aetiology, sEMG activity during eccentric contraction was of most interest. Finally, we estimated the number of previously injured participants necessary to observe a statistically significant between-limb difference in eccentric knee flexor strength and had no way of estimating the size of the differences in hamstring EMG activity in eccentric or concentric actions. Consequently, the power of the study may have been too small to detect between limb differences in medial hamstring EMG. A larger sample size should be a consideration for future work, notwithstanding the difficulty in recruiting athletes for the INJ group.

In conclusion, this study is the first to report that athletes with a history of unilateral HSI display reductions in the sEMG activity of a previously injured BF_L during eccentric contractions and no difference in the median power frequency of either

hamstring head during concentric or eccentric contractions. Furthermore, strength was suppressed during both contraction modes in the injured limb compared to the uninjured limb. Previous HSI may result in between limb alterations in neuromuscular function and rehabilitation practices need to consider the recovery of strength and activation during eccentric contractions as markers of successful rehabilitation as this may assist in reducing the incidence of hamstring strain injury recurrence.

5 STUDY THREE

Rate of torque and electromyographical development during anticipated eccentric contraction is lower in previously strained hamstrings.

This chapter comprises the following paper published in the American Journal of Sports Medicine:

Opar DA, Williams MD, Timmins RG, Dear NM, Shield AJ. Rate of torque and electromyographical development during anticipated eccentric contraction is lower in previously strained hamstrings. American Journal of Sports Medicine. 2013; 41(1):116-25.

The authors listed have certified that:

1. they meet the criteria for authorship in that they have participated in the conception, execution, or interpretation, of at least that part of the publication in their field of expertise;
2. they take public responsibility for their part of the publication, except for the responsible author who accepts overall responsibility for the publication;
3. there are no other authors of the publication according to these criteria;
4. potential conflicts of interest have been disclosed to (a) granting bodies, (b) the editor or publisher of journals or other publications, and (c) the head of the responsible academic unit, and
5. they agree to the use of the publication in the student's thesis and its publication on the QUT ePrints database consistent with any limitations set by publisher requirements.

In the case of this chapter, the following contributions were made:

Contributor	Statement of contribution
David Opar	Determined experimental design, gained ethical approval, participant recruitment, data collection, data analysis, statistical analysis, wrote and submitted the manuscript, responded to reviewer comments approved final proof. Signature: [signature] Date: 13/5/13
Morgan Williams	Determined experimental design, statistical analysis, assisted writing the manuscript, responded to reviewer comments approved final proof.
Ryan Timmins	Participant recruitment, data collection, data analysis.
Nuala Dear	Participant recruitment, data collection, data analysis.
Anthony Shield	Determined experimental design, assisted with ethical approval, assisted writing the manuscript.

Principal Supervisor Confirmation

I have sighted email or other correspondence from all Co-authors confirming their certifying authorship.

ANTHONY SHIELD [signature] 13/5/13

Name Signature Date

5.1 LINKING PARAGRAPH

Chapter 4 examined the impact of prior hamstring strain injury (HSI) on neuromuscular function of the hamstrings during maximum voluntary contractions and this investigation was important in determining that altered neural function is specific to the previously injured muscle and present only during eccentric contractions. These findings allowed the investigation carried out in Chapter 5 to be specific to this contraction mode. The following investigation examined whether previous HSI also impacted upon neuromuscular hamstring function during eccentric contractions that required high rates of torque development (RTD). Measures of neuromuscular function during high RTD tasks may be a more valid measure than using maximum voluntary contractions given the high lengthening velocities of the hamstrings during the injurious terminal swing phase of the running cycle.

5.2 OVERVIEW

HSIs are prevalent in sport and re-injury rates have been high for many years. Whilst much focus has centred on the impact of previous HSI on maximal eccentric strength, high rates of torque development is also of interest, given the important role of the hamstrings during the terminal swing phase of running. The impact of prior strain injury on electromyographical (EMG) activity of the hamstrings during tasks requiring high RTD has received little attention. The purpose of this study was to determine if recreational athletes with a history of unilateral HSI, who have returned to training and competition, will exhibit lower levels of EMG activity during eccentric contraction, RTD and impulse (IMP) 30, 50 and 100ms after the onset of EMG activity or torque development in the previously injured limb compared to the uninjured limb. Twenty-six recreational athletes were recruited. Of these, 13 athletes had a history of unilateral HSI (all confined to biceps femoris long head (BF_L)) and 13 had no history of HSI. Following familiarisation, all athletes undertook isokinetic dynamometry testing and surface electromyography (sEMG) assessment of the BF_L and medial hamstrings (MH) during eccentric contractions at -60 and -180^{o}.s^{-1}. In the injured limb of the injured group, compared to the contralateral uninjured limb RTD and IMP was lower during -60^{o}.s-^{1} eccentric contractions at 50 (RTD, $p = 0.008$, ES = 1.12; IMP, $p = 0.005$, ES = 0.87) and 100ms (RTD, $p = 0.001$, ES = 1.27; IMP, $p < 0.001$, ES = 1.20) after the onset of contraction. BF_L muscle activation was lower at 100ms at both contraction speeds (-60^{o}.s-^{1}, $p = 0.009$, ES = 0.80; -180^{o}.s-^{1}, $p = 0.009$, ES = 0.92). MH activation did not differ between limbs in the injured group. Comparisons in the uninjured group showed no significant between limbs difference for any variables. Previously injured hamstrings displayed lower RTD and IMP

during slow maximal eccentric contraction compared to the contralateral uninjured limb. Lower sEMG activity was confined to the BF_L. Regardless of whether these deficits are the cause of or the result of injury, these findings could have important implications for HSI and re-injury. Particularly, given the importance of high levels of muscle activity to bring about specific muscular adaptations, lower levels of EMG activity may limit the adaptive response to rehabilitation interventions and suggest greater attention be given to neural function of the knee flexors following HSI.

5.3 INTRODUCTION

Muscle strain injuries are problematic for elite, sub-elite and recreational level athletes participating in running based sports.(7, 11, 15, 19) Of all muscle strain injuries in sport, HSIs are the most prevalent. (7, 11, 15, 19) HSIs result in considerable lost time from training and absence from competition, decrements in athlete performance and, in team sports settings, a financial burden for the club or organisation.(142) One of the most prominent consequences of HSIs that is yet to be resolved is the high rates of reinjury, an issue of great importance considering previous HSI is consistently identified as the primary risk factor for future injury.(42) Whilst the existence of this injury-reinjury cycle is acknowledged,(43) success in reducing reinjury rates in one sport has been largely attributed to increased convalescences,(7) more so than due to a greater understanding of the maladaptations associated with previous injury or improved rehabilitation practices.

Scant attention has been given to the potential for unattended neural maladaptations associated with a previous insult to increase the likelihood of future HSI. Recent work has reported lower levels of EMG activity in the previously injured hamstring during maximal voluntary eccentric contractions tested at the movement speed of -$60^{0}.s^{-1}$.(130) That study was the first to provide empirical evidence that lower EMG activity in a previously injured hamstring during maximal eccentric contractions exists. However, many other aspects of neural function are yet to be examined. EMG activity during rapid force generation is one such avenue of further investigation. Such work is warranted given one of the primary roles of the hamstring muscle group is rapid deceleration of the advancing thigh during the terminal swing phase of

high speed running.(37) Optimal hamstring function during this portion of the gait cycle is important as terminal swing is considered by some to be most injurious phase of gait as it combines moderate muscle strains and high force eccentric contraction.(35, 56) As such, high RTD (Δtorque/Δtime) and early contractile IMP (the area under the time vs. torque curve) during eccentric contractions are important characteristics of hamstring function because the limited time available for deceleration (~100ms(55)) prevents the development of maximal torque.(165) Undoubtedly, musculotendinous properties, such as muscle size, relative area of fast-twitch fibers, myosin heavy chain isoform composition and tendon stiffness partly impact on RTD,(166-168) however, the magnitude of EMG activity also contributes. Specifically, the amount of EMG activity during the early phase of the contraction has a positive relationship with RTD.(159, 169) Whether the initial magnitude of EMG activity is less in a previously injured hamstring and whether this results in lower initial eccentric RTD and IMP is, however, yet to be examined.

Measures of RTD, IMP and concurrent EMG activity have been obtained largely during isometric contractions. The information obtained may be limited given the importance of eccentric strength in the aetiology of HSIs. Therefore, assessment of these variables during eccentric contraction may be considered better suited. Yet, the potential to do so is somewhat limited mainly due to the lag between the onset of torque development and the movement of the isokinetic dynamometer lever arm, which we have observed in our laboratory to be in excess of 100ms. To some extent this issue can be overcome through the use of an anticipated eccentric contraction whereby the participant performs an isokinetic eccentric action, however given the

short time frame over which RTD, IMP and EMG activity is analysed the actual contraction is quasi-isometric. Nevertheless, the intention to perform an eccentric action has been shown to result in greater movement related cortical potential compared to concentric actions.(170) This suggests that the execution of motor activity is modulated according to the contraction type to be performed.(170) Indeed, contraction mode specific neural control has been evidenced previously via sEMG with these anticipated eccentric contractions(171) suggesting that contraction mode specific information about EMG activity can be determined with such an experimental design. Therefore, the purpose of the current study was to examine if a previously injured hamstring displayed lower RTD, IMP and concurrent early EMG activity from the BF_L and MH during anticipated slow and fast eccentric actions in comparison to the contralateral uninjured hamstring. sEMG activity was recorded from both BF_L and MH to determine if alterations in EMG activity were confined to the previously injured hamstring muscle. A control group was also examined to demonstrate that limb dominance did not influence RTD, IMP or hamstring EMG activity.

5.4 METHODS

5.4.1 PARTICIPANTS

Recreational level male athletes (n=26) were recruited to participate in the study. All participated in running based sports such as Australian football, soccer, sprinting and touch rugby. Of these, 13 athletes (26.6 ± 5.8 years; 1.8 ± 0.04m; 83.2 ± 14.3kg) had sustained at least one grade II HSI within the last 36 months and another 13 athletes (25.9 ± 3.4 years; 1.8 ± 0.05m; 82.8 ± 7.5 kg) had no history of HSIs. All participants were free of any other lower limb injury, were fully recovered from their previous HSIs and active in their chosen sport at the time of testing. For all athletes limb dominance was defined as the preferred kicking leg. All testing procedures were approved by the University Human Research Ethics Committee. Participants gave informed written consent prior to testing after having all procedures explained to them.

5.4.2 PARTICIPANT INJURY HISTORIES

Following recruitment, participants completed an injury questionnaire with their chosen practitioner (i.e. physiotherapist) who had previously diagnosed and treated all the athletes HSIs. As per previous investigations(130) the notes taken from clinical examination were used to detail the: date of injury and return to pre-injured levels of training and competition; severity (grade I, II or III)(21); location with respect to limb dominance and specific hamstring muscle (BF_L or MH) injured; and rehabilitation details of all previous HSIs. Athletes were considered to be successfully rehabilitated when they returned to pre-injury levels of training and were available for match selection or competition.(157)

5.4.3 sEMG

Myoelectrical activity was measured via sEMG from the MH and BF_L through the use of circular bipolar pre-gelled silver/silver-cholride sEMG electrodes (10mm diameter, 25mm inter-electrode distance). After preparation of the skin via shaving, abrasion and sterilisation, electrodes were placed on the posterior thigh half way between the ischial tuberosity and tibial epicondyles, as per the Surface Electromyography for the Non-Invasive Assessment of Muscles guidelines.(158) Muscle bellies were identified via palpation during forceful isometric knee flexion and correct placement was confirmed by observing sEMG activity during active internal and external rotation of the flexed knee.

5.4.4 ISOKINETIC DYNAMOMETRY

Assessment of knee flexor RTD and IMP was performed on a Biodex Systems 3 Dynamometer (Biodex Medical Systems, Shirley, NY). Participants were seated on a custom pad, placed on top of the original seat, which contained two holes at the level of the posterior mid thigh to minimise movement artefact from sEMG electrodes on the dynamometer seat. The hips were flexed at 85^o from neutral with the lateral epicondyle of the femur carefully aligned with the fulcrum of the dynamometer. The tested leg was attached to the lever of the dynamometer via a Velcro strap and padded restraints were fastened across the trunk, hips and mid thigh of the tested leg to isolate movement to the knee joint. The range of motion was set at 5-90^o of knee flexion (0^o = full knee extension; knee joint angle at start position = 90^o) and correction for limb weight was performed throughout the range of motion. Three sets of four submaximal concentric contractions of the knee extensors and flexors were

performed at +240^{o}.s^{-1} as a warm-up to prepare the participant for maximal effort in the following sets. Eccentric testing for both legs consisted of three sets of three consecutive eccentric maximum voluntary contractions of the knee flexors at speeds of -60^{o}.s^{-1} and -180^{o}.s^{-1} with 30 seconds rest between sets. The leg and speed testing orders were randomised and athletes were informed of the testing speed prior to each set. Athletes were instructed to remain relaxed prior to contraction to allow a stable baseline measurement of torque and sEMG to be obtained. Athletes were instructed to push their heel back as quickly as they could towards their gluteus when given the signal to contract and were encouraged verbally by the investigators to ensure maximal effort. The signal to contract was delivered verbally by the investigators. All athletes were required to attend at least one familiarisation session and one testing session with ≥ seven days between each session.

5.4.5 DATA ANALYSIS

For each movement speed the three contractions with the highest peak torque were used for further analysis. Dynamometer torque and lever position data were transferred to a personal computer at 1 kHz and stored for later analysis. RTD was determined as the mean of the average slope of the torque-time trace (Δtorque/Δtime) for the three selected repetitions from the onset of contraction through until 30, 50 and 100ms of the contraction. Onset of contraction was defined as when torque deviated 4Nm from the baseline level of torque at rest (Figure 5-1).(172) IMP was calculated as the area under the torque-time trace across the same time periods. sEMG data was sampled simultaneously with dynamometer data at 1kHz through a 16-bit PowerLab26T AD recording unit with in-built anti-aliasing filter

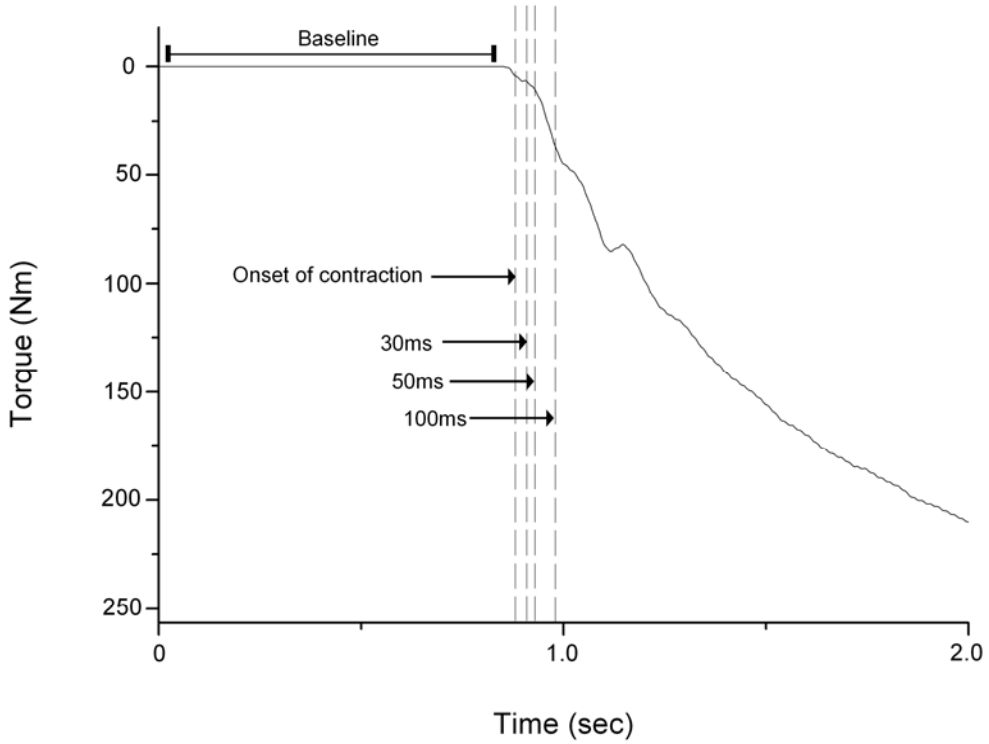


Figure 5-1. Representative torque-time trace. Prior to the onset of contraction baseline levels of torque were determined. Onset of contraction was defined as when knee flexor torque deviated by 4.0Nm from baseline. Rate of torque development was determined as the average change in torque over time (Δtorque/Δtime) at 30, 50, 100ms from onset of contraction development.

(ADInstruments, New South Wales, Australia) (amplification = 1000; common mode rejection ratio = 110dB; Input impedance = 100 MΩ; fixed gain) and stored for later analysis where it was fourth order Butterworth filtered between 20-500Hz (24dB roll off) using MATLAB (MathWorks, Natick, Massachusetts) and then full wave rectified using the root-mean-square method. For each contraction, sEMG data for MH and BF was normalised to the maximum magnitude of the rectified sEMG signal for that contraction, for each muscle respectively. sEMG activity was defined as the

area under the rectified sEMG-time trace, commonly referred to as integrated EMG, and was measured across 30, 50 and 100ms after the onset of myoelectrical activity. Onset of sEMG activity was determined by smoothing the rectified EMG signal (100 point moving average) and then identifying when the smoothed rectified signal rose above 10% of the maximum signal for the final time.(115) The identification of onset was then confirmed by visual examination of the raw and rectified (unsmoothed) sEMG signal at the same time point. All analysis was performed using LabCart 7.3 (ADInstruments, New South Wales, Australia).

5.4.6 STATISTICAL ANALYSIS

Data was analysed using JMP version 9.0 Pro Statistical Discovery Software (SAS Inc). Aligned with the study's primary objectives, comparisons were made for each dependent variable (RTD, IMP and BF and MH sEMG activity) between the injured and uninjured limbs in the injured group. Comparisons between dominant and non-dominant limbs in the uninjured group were also made to determine any influence of limb dominance. The use of ANOVA models was deemed not valid as assumptions were not met for the homogeneity of variance for the dependent variables ($p < 0.05$).(173) As such, dependent variables were compared using two tailed paired t tests for both groups. Bonferroni corrections were performed to account for three comparisons made for each dependent variable across the velocities used, with significance adjusted to $p<0.0167$. To assess the magnitudes of the differences Cohen's d was also used to report effect size (ES).

5.5 RESULTS

5.5.1 PARTICIPANTS

The two groups were similar with respect to age, height and body mass (Injured group, 26.6 ± 5.8 years; 1.8 ± 0.04m; 83.2 ± 14.3kg; Uninjured group, 25.9 ± 3.4 years; 1.8 ± 0.05m; 82.8 ± 7.5 kg). All athletes from the injured group had suffered at least one grade II HSI in the last 36 months. The total number of HSIs sustained by each athlete in the injured group ranged between one and four (median = 2) in the same 36 month period. All injuries were confined to the BF_L. Median time since most recent HSI was 3.9 months (range = 1.0 – 18.2), with median time taken to return to pre-injured levels of competition being 4 weeks (range = 2 - 6). All athletes from the injured group reported standard rehabilitation progression (i.e. Ref (131)) guided by their physiotherapist, with all but one of the injured athletes reporting some eccentric conditioning as part of their late phase rehabilitation program.

5.5.2 RTD AND IMP

RTD and IMP was significantly lower in the previously injured knee flexor for -$60^{o}.s^{-1}$ anticipated eccentric contractions at 50ms (RTD, p=0.008, ES=1.12; IMP, p=0.005, ES=0.87) and 100ms (RTD, p=0.001, ES=1.27; IMP, p<0.001, ES=1.20) after the onset of contraction (Figure 5-2, 5-3; Table 5-1). There was no significant difference for RTD or IMP during anticipated eccentric contractions at -$180^{o}.s^{-1}$ at any time point (Figure 5-2, 5-3; Table 5-1). There were no between limb differences for either variable in the control group (Figure 5-4, 5-5; Table 5-2).

5.5.3 sEMG

With respect to myoelectrical activity of BF_L, normalised iEMG was lower at 100ms at both contraction speeds in the previously injured limbs in the injured group (-60^o.s-^{1}, p=0.009, ES=0.80; -180^o.s-^{1}, p=0.009, ES=0.92) (Figure 5-6; Table 5-3), but there were no significant differences between limbs in the control group (Figure 5-8; Table 5-4). No differences existed with respect to MH iEMG in either group (Figure 5-7, 5-9; Table 5-3, 5-4).

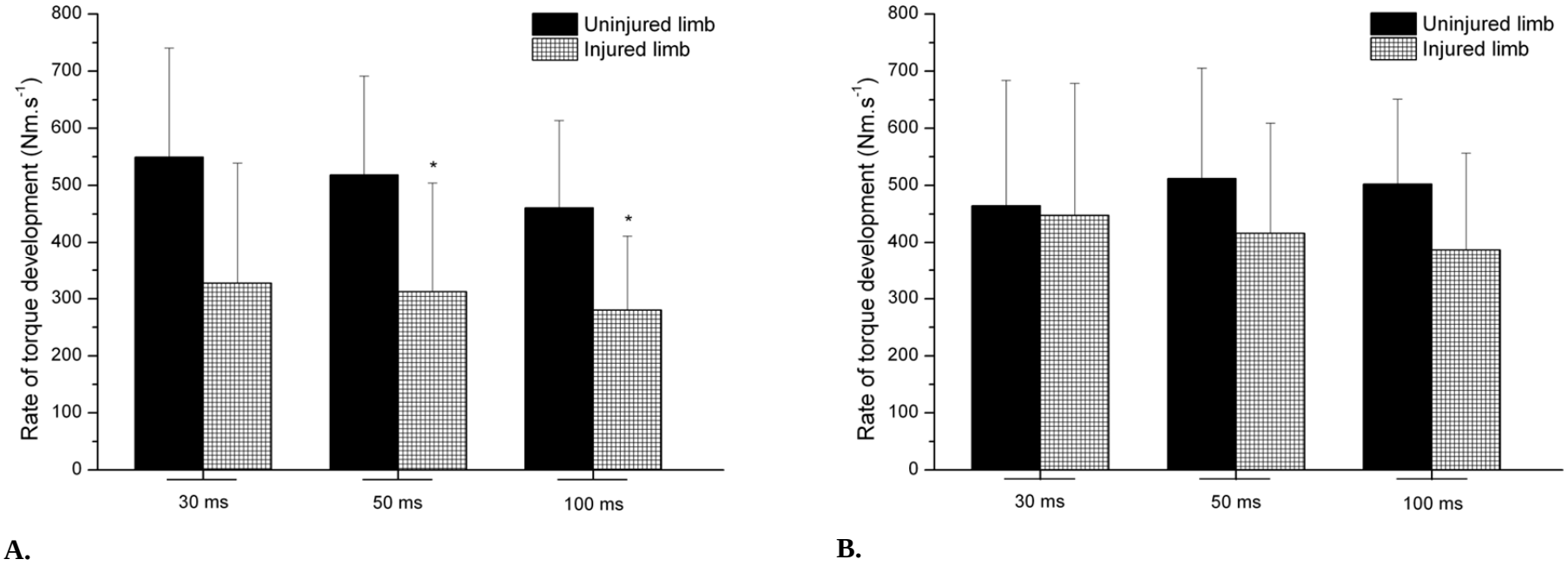


Figure 5-2. Comparisons between the uninjured and injured limbs of previously injured athletes of knee flexor rate of torque development during anticipated eccentric contractions at A) $-60^{o}.s^{-1}$ and B) $-180^{o}.s^{-1}$ 30, 50 and 100ms from the onset of torque development. Error bars indicate standard deviation. *$p<0.0167$ uninjured vs. injured limbs.

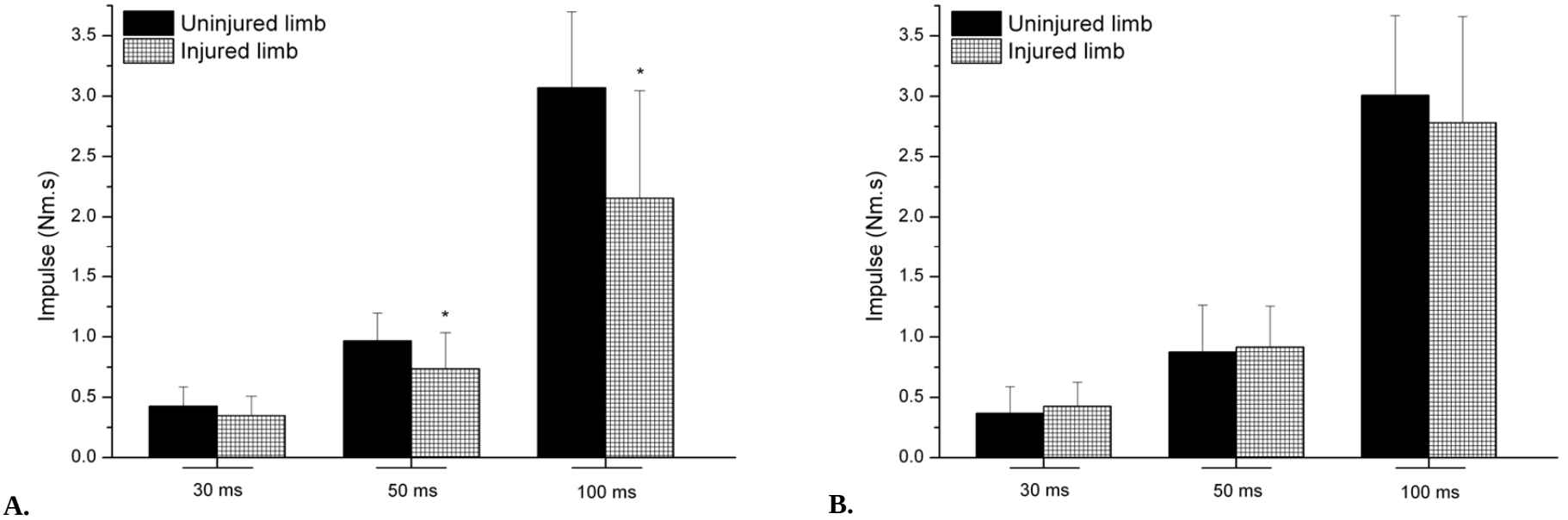


Figure 5-3. Comparisons between the uninjured and injured limbs of previously injured athletes of knee flexor impulse at A) $-60^{o}.s^{-1}$ and B) $-180^{o}.s^{-1}$ at 30, 50 and 100ms from the onset of torque development. Error bars indicate standard deviation. *$p<0.0167$ uninjured vs. injured limbs.

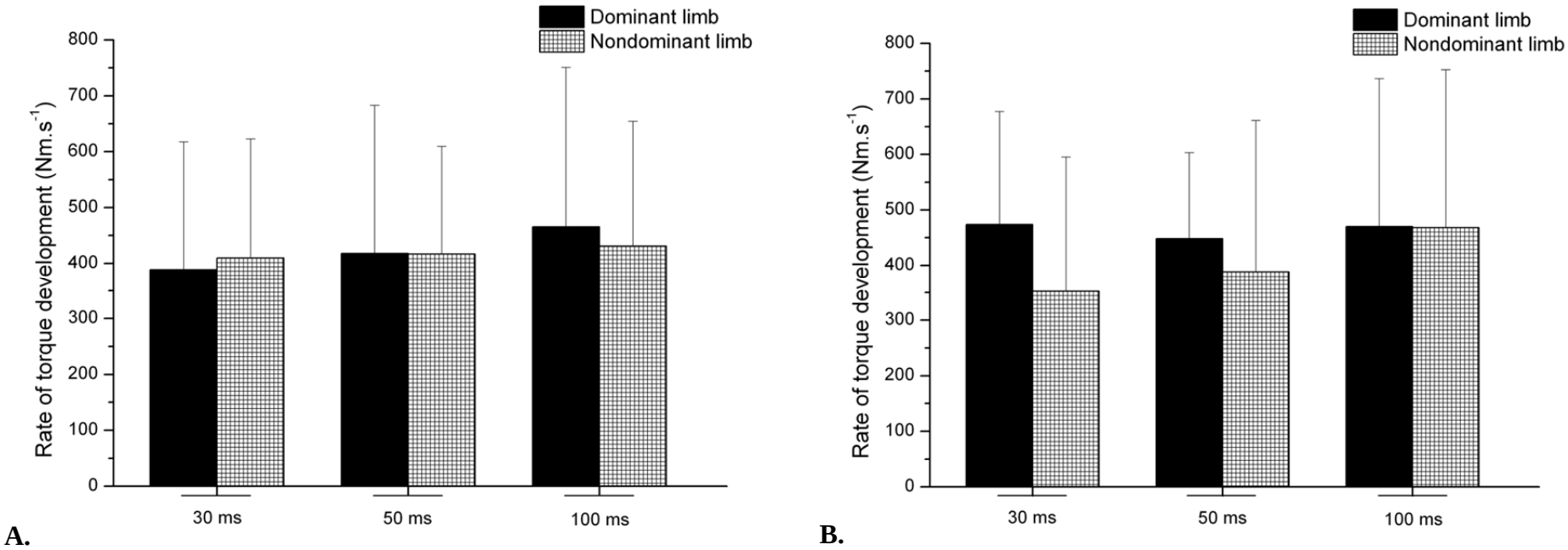


Figure 5-4. Comparisons between the dominant and nondominant limbs of uninjured athletes of knee flexor rate of torque development at A) -60°.s^{-1} and B) -180°.s^{-1} at 30, 50 and 100ms from the onset of torque development. Error bars indicate standard deviation.

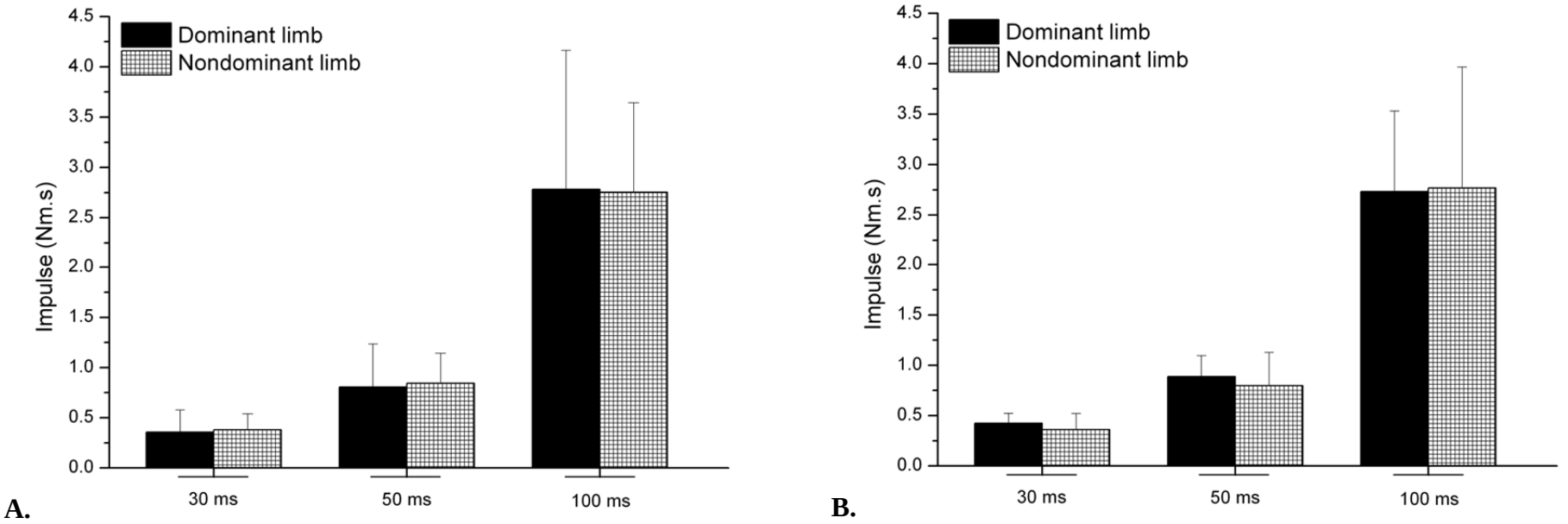


Figure 5-5. Comparisons between the dominant and nondominant limbs of uninjured athletes of knee flexor impulse at A) $-60^{o}.s^{-1}$ and B) $-180^{o}.s^{-1}$ at 30, 50 and 100ms from the onset of torque development. Error bars indicate standard deviation.

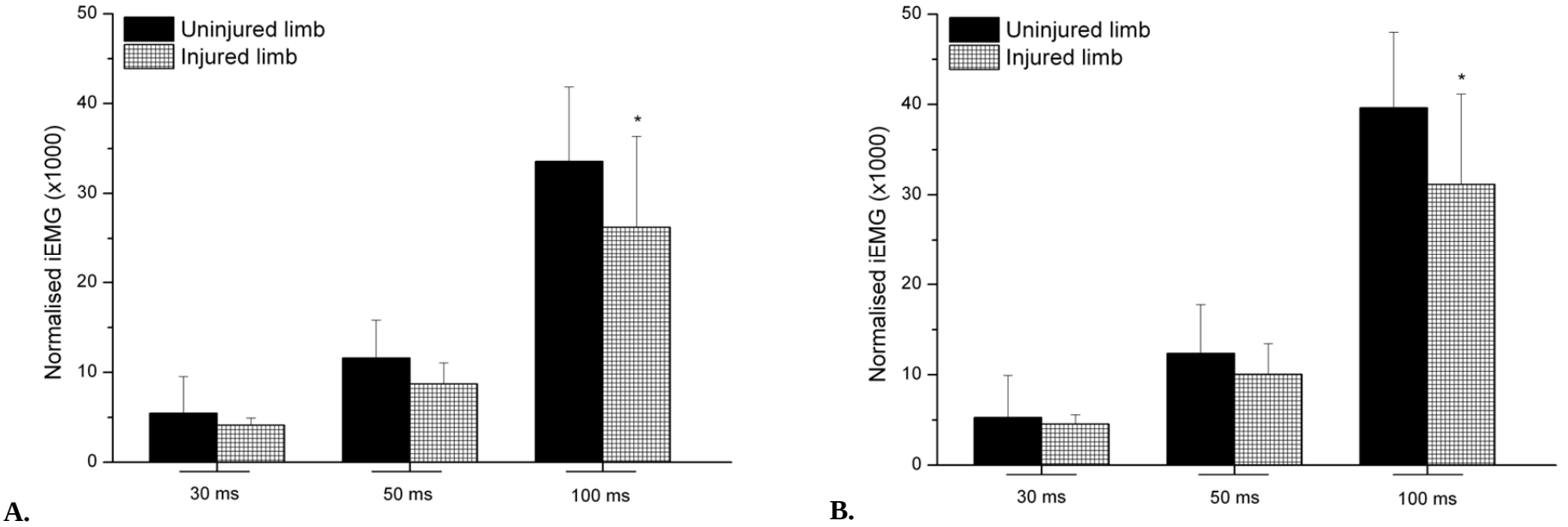


Figure 5-6. Comparisons between the uninjured and injured limbs of previously injured athletes of integrated electromyography (iEMG) from the biceps femoris long head at A) $-60^{o}.s^{-1}$ and B) $-180^{o}.s^{-1}$ at 30, 50 and 100ms from the onset of electromyographical activity. Error bars indicate standard deviation. *$p<0.0167$ uninjured vs. injured limbs.

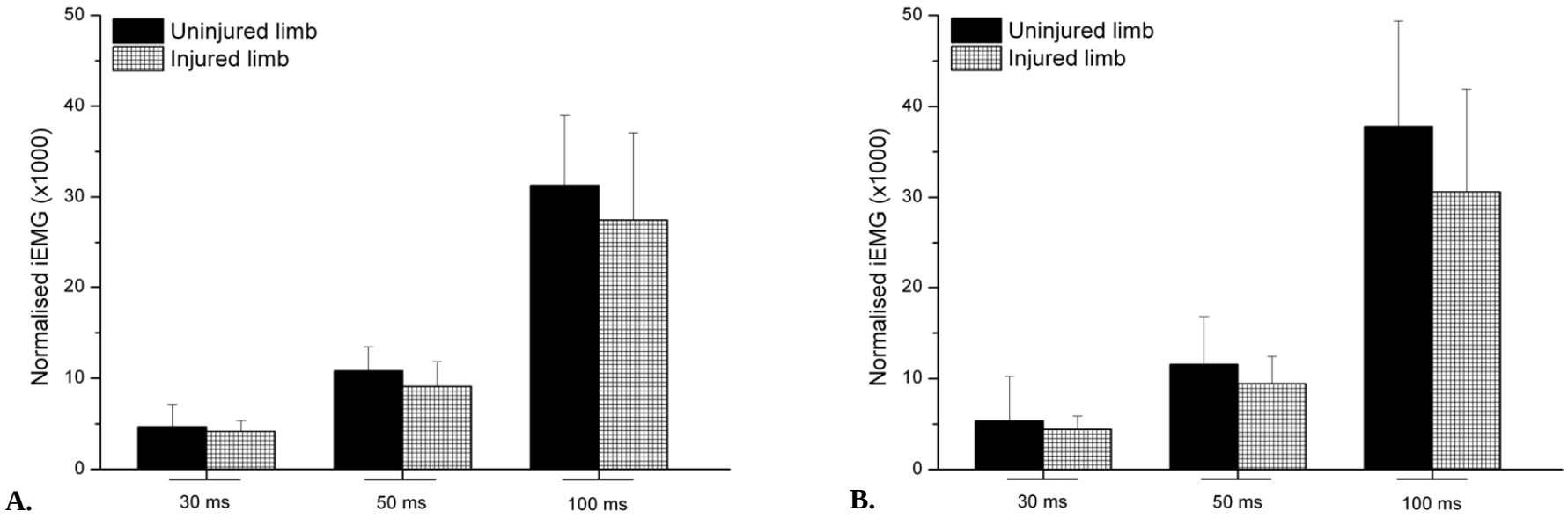


Figure 5-7. Comparisons between the uninjured and injured limbs of previously injured athletes of integrated electromyography (iEMG) from the medial hamstrings at C) $-60^{o}.s^{-1}$ and D) $-180^{o}.s^{-1}$ at 30, 50 and 100ms from the onset of electromyographical activity. Error bars indicate standard deviation.

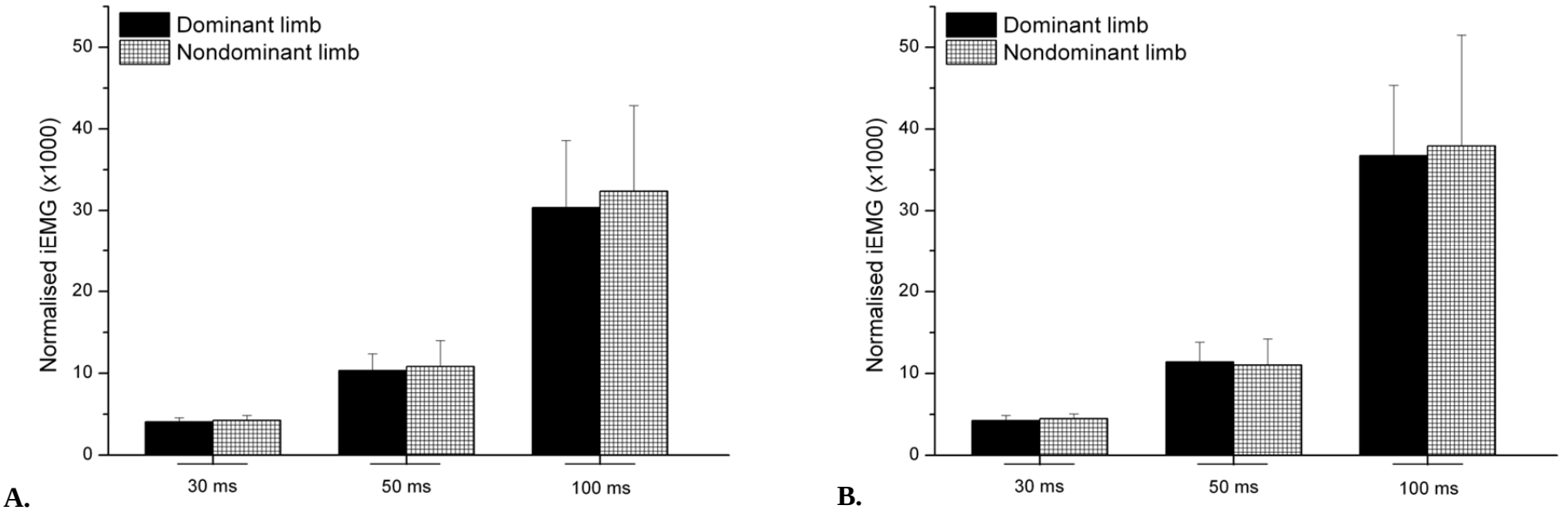


Figure 5-8. Comparisons between the dominant and nondominant limbs of uninjured athletes of integrated electromyography (iEMG) from the biceps femoris long head at A) $-60^{o}.s^{-1}$ and B) $-180^{o}.s^{-1}$ at 30, 50 and 100ms from the onset of electromyographical activity. Error bars indicate standard deviation.

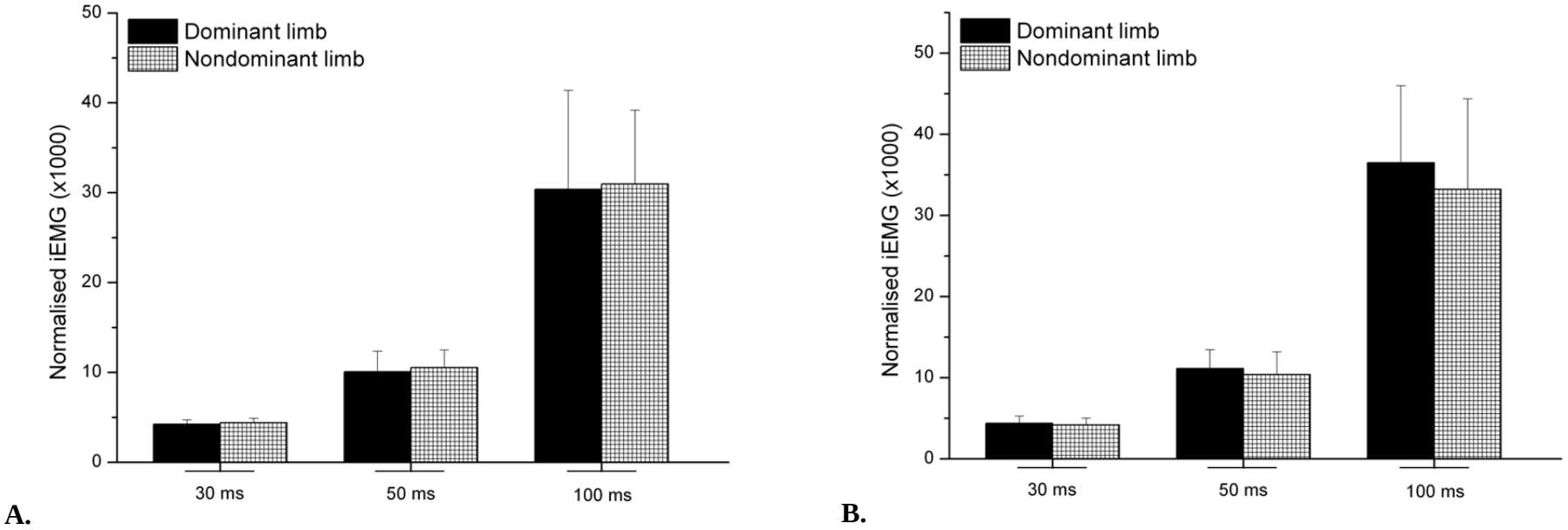


Figure 5-9. Comparisons between the dominant and nondominant limbs of uninjured athletes of integrated electromyography (iEMG) from the medial hamstrings at C) $-60^{o}.s^{-1}$ and D) $-180^{o}.s^{-1}$ at 30, 50 and 100ms from the onset of electromyographical activity. Error bars indicate standard deviation.

Table 5-1. The rate of torque development and impulse of the knee flexors during anticipated eccentric contractions at 30, 50 and 100ms after the onset of torque development in athletes with a history of unilateral hamstring strain injury.

Movement velocity		Injured group							
		Rate of torque development ($Nm.s^{-1}$)				Impulse (Nm.s)			
		Injured limb	Uninjured limb	P	ES	Injured limb	Uninjured limb	P	ES
-60									
	30ms	327.41 (± 211.31)	549.36 (± 191.05)	0.0320	1.10	0.35 (± 0.16)	0.43 (± 0.16)	0.0640	0.49
	50ms	312.27 (± 191.78)	518.54 (± 172.81)	0.0080*	1.13	0.73 (± 0.30)	0.97 (± 0.23)	0.0050*	0.87
	100ms	280.03 (± 131.42)	460.54 (± 152.94)	0.0010*	1.27	2.15 (± 0.89)	3.07 (± 0.63)	<0.0010*	1.20
-180									
	30ms	447.46 (± 231.54)	464.21 (± 219.98)	0.8730	0.07	0.43 (± 0.20)	0.37 (± 0.22)	0.6020	0.27
	50ms	416.04 (± 193.02)	511.88 (± 193.53)	0.2840	0.50	0.92 (± 0.34)	0.87 (± 0.39)	0.2320	0.11
	100ms	386.79 (± 169.85)	502.33 (± 148.61)	0.0640	0.73	2.78 (± 0.88)	3.01 (± 0.66)	0.0900	0.30

RTD, rate of torque development; IMP, impulse; ES, Cohen's d effect size. Data are presented as mean (± standard deviation). Significance was set at $p < 0.0167$. * indicates significant difference between injured and uninjured limbs.

Table 5-2. The rate of torque development and impulse of the knee flexors during anticipated eccentric contractions at 30, 50 and 100ms after the onset of torque development in athletes without a history of unilateral hamstring strain injury.

Movement velocity		Uninjured group							
		Rate of torque development ($Nm.s^{-1}$)				Impulse (Nm.s)			
		Dominant limb	Non-dominant limb	P	ES	Dominant limb	Non-dominant	P	ES
-60									
	30ms	388.75 (± 228.79)	409.85 (± 213.05)	0.7730	0.10	0.36 (± 0.22)	0.38 (± 0.16)	0.6530	0.12
	50ms	417.79 (± 265.17)	416.74 (± 192.94)	0.9870	0.00	0.81 (± 0.43)	0.84 (± 0.30)	0.6830	0.10
	100ms	465.49 (± 285.45)	430.88 (± 223.38)	0.4370	0.14	2.78 (± 1.38)	2.75 (± 0.89)	0.9000	0.03
-180									
	30ms	473.95 (± 203.37)	352.42 (± 243.00)	0.1880	0.54	0.42 (± 0.10)	0.36 (± 0.16)	0.1640	0.47
	50ms	448.12 (± 154.98)	388.41 (± 273.04)	0.3870	0.28	0.89 (± 0.21)	0.80 (± 0.33)	0.3570	0.33
	100ms	470.05 (± 266.72)	468.29 (± 284.54)	0.9470	0.01	2.73 (± 0.80)	2.77 (± 1.20)	0.9130	0.04

RTD, rate of torque development; IMP, impulse; ES, Cohen's d effect size. Data are presented as mean (± standard deviation).

Table 5-3. The normalised iEMG activity (x 1000) of biceps femoris long head and medial hamstrings during anticipated eccentric contractions at 30, 50 and 100ms after the onset of myoelectrical activity in athletes with a history of unilateral hamstring strain injury.

Movement velocity		Injured group							
		Normalised iEMG BF_L activity (x1000)				Normalised iEMG MH activity (x1000)			
		Injured limb	Uninjured limb	P	ES	Injured limb	Uninjured limb	P	ES
-60									
	30ms	4.13 (± 0.79)	5.45 (± 4.09)	0.2690	0.54	4.18 (± 1.19)	4.69 (± 2.45)	0.5680	0.28
	50ms	8.71 (± 2.35)	11.61 (± 4.19)	0.0300	0.88	9.11 (± 2.72)	10.82 (± 2.66)	0.1120	0.63
	100ms	6.25 (± 10.11)	33.57 (± 8.29)	0.0090*	0.80	27.47 (± 9.62)	31.29 (± 7.73)	0.1480	0.44
-180									
	30ms	4.54 (± 1.04)	5.28 (± 4.65)	0.5930	0.26	4.42 (± 1.47)	5.36 (± 4.91)	0.4980	0.30
	50ms	10.04 (± 3.41)	12.38 (± 5.36)	0.1220	0.53	9.43 (± 3.02)	11.54 (± 5.25)	0.2060	0.51
	100ms	31.16 (± 10.01)	39.64 (± 8.36)	0.0090*	0.92	30.60 (± 11.31)	37.82 (± 11.57)	0.1360	0.63

iEMG, integrated surface electromyography; BF_L, biceps femoris long head; MH, medial hamstrings; ES, Cohen's d effect size. Data are presented as mean (± standard error of the mean). Significance was set at $p < 0.0167$. * indicates significant difference between injured and uninjured limbs.

Table 5-4. The normalised iEMG activity (x 1000) of biceps femoris long head and medial hamstrings during anticipated eccentric contractions at 30, 50 and 100ms after the onset of myoelectrical activity in athletes without a history of unilateral hamstring strain injury.

Movement velocity ($^{o}.s^{-1}$)	Uninjured group							
	Normalised iEMG BF_L activity (x1000)				Normalised iEMG MH activity (x1000)			
	Dominant limb	Non-dominant	P	ES	Dominant limb	Non-dominant	P	ES
-60								
30ms	4.07 (± 0.49)	4.23 (± 0.60)	0.5390	0.30	4.25 (± 0.48)	4.41 (± 0.49)	0.3340	0.33
50ms	10.32(± 2.05)	10.83 (± 3.18)	0.4320	0.20	10.08 (± 2.28)	10.55 (± 1.96)	0.4200	0.22
100ms	30.35 (± 8.22)	32.36 (± 10.50)	0.3110	0.21	30.38 (± 11.03)	30.97 (± 8.23)	0.8630	0.06
-180								
30ms	4.25 (± 0.64)	4.48 (± 0.58)	0.3370	0.37	4.38 (± 0.90)	4.18 (± 0.83)	0.5360	0.23
50ms	11.42 (± 2.39)	11.03 (± 3.19)	0.5730	0.14	11.12 (± 2.34)	10.38 (± 2.81)	0.4130	0.29
100ms	36.75 (± 8.61)	37.93 (± 13.58)	0.7140	0.11	36.48 (± 9.51)	33.23 (± 11.15)	0.2450	0.31

iEMG, integrated surface electromyography; BF_L, biceps femoris long head; MH, medial hamstrings; ES, Cohen's d effect size. Data are presented as mean (± standard error of the mean).

5.6 DISCUSSION

The hamstring muscle group is the most commonly strained muscle in running based sports. (7, 11, 15, 19) This is purportedly due to the demands of high speed running and specifically the need for rapid deceleration of the flexing hip and extending knee during terminal swing.(35, 56) As such the ability of the biarticular hamstrings to generate eccentric force rapidly is a key feature of hamstring function. The current study examined whether athletes with a prior unilateral HSI history displayed lower levels of RTD, IMP and EMG activity in the previously injured hamstring compared to the contralateral uninjured hamstring for brief periods following the onset of anticipated eccentric contractions. The novel findings from this study are that recreational athletes with a history of HSIs confined to the BF_L exhibited i) lesser RTD and IMP 50ms and 100ms after the onset of an anticipated eccentric contractions at $-60^0.s^{-1}$ and; ii) lesser BF_L EMG activity at 100ms after the onset of EMG activity in anticipation of eccentric contractions at $-60^0.s^{-1}$ and $-180^0.s^{-1}$ in the previously injured limb compared to the uninjured limb. Of further importance was that EMG activity of the MH was not different between limbs in the injured group. There were also no differences found between dominant and non-dominant limb for RTD, IMP or EMG activity in the control group, indicating no influence of limb dominance.

This is, to our knowledge, the first study to examine RTD, IMP and concurrent EMG activity in previously injured hamstrings, which makes comparisons to previous work difficult. One previous study has examined the impact of a simulated handball game on isometric knee flexor function and this study reported higher baseline RTD

relative to bodyweight (6.92 – 9.27Nm/s/kg) compared to the uninjured limbs (4.82 – 5.41Nm/s/kg) in the current study.(172) The divergent RTD findings may be explained by the methodological differences such as athlete expertise (recreational active vs. elite handball players), different knee joint angles used to assess RTD (90^{o} vs. 70^{o} of knee flexion) and the use of anticipated eccentric contraction as opposed to isometric RTD in previous work.

The finding that a previous strain injury to BF_L results in a lesser ability to generate torque quickly in anticipation of an eccentric contraction may have important consequences for recurrent HSI risk and current rehabilitation practices. This is because the time frame in which the knee flexors have to decelerate the flexing hip and extending knee joints during terminal swing is limited (~100ms(55)). As such the rapid development of eccentric torque is paramount to minimise the risk of overlengthening of the hamstrings. If, as was observed in the current cohort, previously injured limbs display lower knee flexor RTD and IMP and lower BF_L EMG activity up to 100ms following the onset of contraction it might be expected to increase the work required of the hamstrings at terminal swing to slow the forward moving shank due to poor deceleration during early swing. Furthermore, a lesser ability to produce a decelerating force for a brief period following the onset of contraction would likely increase the work required of the hamstrings at longer muscle lengths and the impact of this may be two fold. Firstly, the increase in work may induce the onset of fatigue earlier in the BF_L, which is the primary knee flexor at long muscle lengths.(124) Given fatigue reduces the amount of energy that can be absorbed by a lengthening muscle(59) this may increase the potential for strain

induced muscle failure. Secondly, unpublished observations from our lab suggest that athletes with a previous HSI to BF_L display lower BF_L EMG activity during eccentric contractions at long lengths. If there are extra demands placed on the BF_L at terminal swing due to poor RTD and IMP, but due to restricted EMG activity at this muscle length the muscle cannot meet these demands then this has the potential to increase the likelihood for hamstring overlengthening. Such overlengthening can be problematic as it may increase the risk of the hamstrings exceeding their mechanical limits(142) or accumulating microscopic muscle damage(76) and this increases the potential for injury/reinjury.

The observations that RTD and IMP were lower in anticipation of a slow, but not fast, eccentric contraction is intriguing given that the EMG activity of the previously injured BF_L was lower in anticipation of both velocities of eccentric contraction. Whilst RTD was not lower in the previously injured limb compared to the contralateral uninjured limb at any time point at $-180^0.s^{-1}$ there was a medium effect size at 100ms following the onset of contraction ($p=0.064$, Cohen's d ES=0.57) and a larger sample may have revealed a significant difference. However, this finding might also be indicative of alterations in coordination of the knee flexor muscles in anticipation of a fast eccentric contraction. Altered coordination may be driven by the intent to protect the previously injured BF_L in anticipation of a high speed eccentric action. In the case of this study other knee flexors, not examined, might be recruited more heavily thus increasing their contribution to knee flexion torque generation, with the most suited candidate being the uniarticular biceps femoris short head (BF_S). Indirect evidence supports this change in contribution to knee flexion

torque, given that a previously injured leg displays compensatory hypertrophy of this muscle,(46) which would be suggestive of an increased volume of work during habitual activities. Moreover, BF_L atrophy has been found,(46) as a possible consequence of reduced activation and disuse following HSI. Whether such a reorganisation of muscle activity exists is, however, yet to be explored and should be an area for future examination.

If significant neuromuscular inhibition of BF exists its benefits are most likely to be confined to the early phase of recovery and rehabilitation. A novel framework proposed previously hypothesises that pain associated with HSI results in prolonged neural deficits which compromise the rehabilitation process.(142) This framework focuses largely on chronic reductions in voluntary activation of the previously injured hamstrings during eccentric contractions and the impact of such a neurological deficit on muscular adaptations (for a thorough discussion of this see Ref (142)). However, reductions in early neural drive of the previously injured BF_L in response to strain injury may present another problematic maladaptation associated with previous HSI. Acute restriction of early neural drive following injury presumably constitutes a strategy to unload the damaged tissue and reduce pain in the acute recovery period.(142) However, chronic reductions in early neural drive would be expected to compromise the rehabilitation process, given the need for high levels of activation to bring about muscular adaptations.(142) The reduction in early EMG activity of BF_L, combined with the restriction of EMG activity of BF_L during maximal eccentric contraction (unpublished observations from our lab), might be expected to reduce the stimulus the previously injured BF_L is exposed to, resulting in

limited muscle hypertrophy and sarcomerogenesis. Decrements in these two factors would be expected to reduce strength and reduce the optimum length of the hamstring muscle group, respectively, and both have been implicated in HSI aetiology.(32, 76) Whilst much work has been done on the contractile and structural(58, 174) implications of strain injury, neural maladaptation and associated changes have been largely neglected and should be the focus of future investigations.

If lower BF_L EMG activity is in response to, and not the cause of, HSI the underpinning mechanism responsible is of interest. At present most studies have examined the impact of resistance training on neural factors that influence RTD. These studies all have focused on mechanisms to explain improved RTD including: increased neural drive; increased motor unit discharge rates; increased motor unit synchronisation; and earlier recruitment of motor units.(175-178) Whether all of these adaptations occur 'in reverse' following HSI remains to be seen, however the current study found that lower EMG activity occurred in the previously injured BF_L. Yet, as the stimulus for neural maladaptation to HSI is hypothesised to be due to pain(142) (as opposed to heavily load or explosive resistance exercise(169)) the altered function of the nervous system may differ markedly. HSIs induce acute(20) and chronic(43, 44) pain particularly in athletes with recurrent strain injuries. Acute muscle pain is known to result in short term neural responses resulting in reduced strength, agonistic activation and muscle endurance, increased antagonistic activity and altered coordination patterns during static and dynamic motor tasks.(179-182) This muscular pain also has the potential to alter central nervous function at both the spinal and supraspinal level, resulting in increased pain sensitivity and an expanded

neuron population of the painful muscle in the dorsal horn of the spinal cord.(156) Pain has the potential to modulate descending neural pathways(183) and by extension the ability to fully activate the motor neuron pool. This maladaptation of neural function might therefore be expected to result in a restriction of EMG activity during the onset of contraction and may be specifically confined to the muscle responsible for the noxious stimulus.

There are some limitations associated with the current work. Firstly, as discussed earlier, the statistical power of the current study was too low to detect small to moderate effect sizes (Cohen's d = 0.2-0.8). A larger sample size might have revealed significant differences between dependent variables that were not identified in the current study. As such a larger sample, also considering the inclusion of female athletes, should be a consideration for future investigations; notwithstanding the difficulty in recruiting athletes for the injured group. The retrospective nature of these findings do not allow for the determination of whether lower levels of RTD, IMP and concurrent early EMG activity of BF_L are the cause of, or the result of HSI. Potentially the lesser EMG activity in the previously injured BF_L could indicate incomplete rehabilitation, whereby the deficits could be ameliorated with further intervention; a permanent lessening of EMG activity in response to injury; or a deficit that was present prior to injury. Future work should investigate whether lower EMG activity, particularly of BF_L, is a risk factor for future HSI and explore what interventions are successful at restoring EMG activity following HSI. Furthermore, we were unable to control the rehabilitation programmes of the current cohort, however all reported largely conventional rehabilitation progression guided by a

physiotherapist. We were also limited because current methodologies do not allow for the performance of eccentric isokinetic knee flexion in such brief time periods as examined in the current study. As such the muscle action performed during the assessed time periods was quasi-isometric. Regardless, the intention to perform an eccentric muscle action results in different cortical(170) and sEMG(171) activity compared to concentric contractions even when performing quasi-isometric contraction.(171) This suggests that information about contraction mode specific EMG activity can be derived from quasi-isometric contractions with the intent of performing an eccentric action. Finally, the use of isokinetic dynamometry at speeds of -60 and $-180^{0}.s^{-1}$ to assess eccentric neuromuscular function is not wholly reflective of the demands placed on the hamstrings during injurious activities such as running and kicking, where greater angular velocities are experienced. The impact of previous HSI on neuromuscular function during these tasks should be examined further. Nevertheless, isokinetic testing combined with sEMG allows for the determination of RTD, IMP and EMG activity whilst controlling for different movement velocities, a variable which was found to influence RTD and IMP in the current study.

In conclusion, we have shown for the first time, to our knowledge, that a previously strained hamstring, which has been rehabilitated sufficiently to return to training and competition, displays lower levels of RTD and IMP in anticipation of a slow maximal eccentric contraction compared to the contralateral uninjured limb. Furthermore, lower early EMG activity was observed in the injured BF_L compared to the contralateral uninjured BF_L in anticipation of fast and slow maximal eccentric

contraction. Regardless of whether these deficits are a response to or the result of muscle strain injury they could have important implications for current preventative and rehabilitation practices. Particularly, given the importance of high levels of muscle activity to bring about specific muscular adaptations, lower levels of EMG activity may limit the adaptive response to rehabilitation interventions. This would be expected to limit the effectiveness of rehabilitation exercises and suggests that consideration be given to deficits in EMG activity following HSI. A greater appreciation for impaired neural function following HSI might be expected to improve rehabilitation outcomes.

6 STUDY FOUR

A novel device using the Nordic hamstring exercise to assess eccentric knee flexor strength: a reliability and retrospective injury study.

This chapter comprises the following manuscript currently under review at the British Journal of Sports Medicine:

Opar DA, Piatkowski T, Williams MD, Shield AJ. A novel device using the Nordic hamstring exercise to assess eccentric knee flexor strength: a reliability and retrospective injury study. British Journal of Sports Medicine. In review.

The authors listed have certified that:

1. they meet the criteria for authorship in that they have participated in the conception, execution, or interpretation, of at least that part of the publication in their field of expertise;
2. they take public responsibility for their part of the publication, except for the responsible author who accepts overall responsibility for the publication;
3. there are no other authors of the publication according to these criteria;
4. potential conflicts of interest have been disclosed to (a) granting bodies, (b) the editor or publisher of journals or other publications, and (c) the head of the responsible academic unit, and
5. they agree to the use of the publication in the student's thesis and its publication on the QUT ePrints database consistent with any limitations set by publisher requirements.

In the case of this chapter, the following contributions were made:

Contributor	Statement of contribution
David Opar	Determined experimental design, gained ethical approval, participant recruitment, data collection, data analysis, statistical analysis, wrote and submitted the manuscript, responded to reviewer comments approved final proof. Signature: [signature] Date: 13/5/13
Timothy Piatkowski	Participant recruitment, data collection, data analysis, statistical
Morgan Williams	Determined experimental design, statistical analysis, assisted writing the manuscript, responded to reviewer comments approved final proof.
Anthony Shield	Determined experimental design, assisted with ethical approval, assisted writing the manuscript.

Principal Supervisor Confirmation

I have sighted email or other correspondence from all Co-authors confirming their certifying authorship.

ANTHONY SHIELD A. Shield 13/5/13

Name Signature Date

6.1 LINKING PARAGRAPH

The preceding three chapters (Chapters 3-5) have identified that fatigue induced by intermittent running and previous hamstring strain injury (HSI) both impact upon eccentric hamstring neuromuscular function, more so than concentric function. However, access to a methodology to assess this strength quality is difficult and involves the use of specialised laboratory-based equipment or field based techniques which have a number of limitations. This chapter explores the reliability of a novel experimental device designed to measure eccentric hamstring strength in the field. Furthermore, the device is then used to measure between limb strength imbalances in elite athletes with a unilateral HSI within the last 12 months and to compare eccentric hamstring strength in injured athletes to uninjured control subjects.

6.2 OVERVIEW

HSIs and re-injuries are the most common injury in sport. Eccentric knee flexor weakness and between limb eccentric strength asymmetry are a major modifiable risk factors for future HSI, however there is a lack of accessible methodologies to assess this strength quality. The aims of this study were 1) to determine if a novel device, designed to measure eccentric knee flexor strength via the Nordic hamstring exercise (NHE), displays acceptable test-retest reliability; 2) to determine normative values for eccentric knee flexor strength derived from the experimental device in individuals without a history of HSI and; 3) to determine if the device could detect weakness in elite athletes with a previous history of unilateral HSI. Thirty recreationally active males without a history of HSI completed NHEs and had their strength measured on the device on two separate occasions. Intraclass correlation coefficients (ICC), typical error (TE) and typical error as a co-efficient of variation (%TE) were established. Normative strength data was determined using the most reliable measurement. An additional 30 elite athletes with a unilateral history of HSI within the last 12 months performed NHEs on the device to determine if eccentric weakness existed. Thirty recreationally active males without a history of HSI completed NHEs and had their strength measured on the device on two separate occasions. Intraclass correlation coefficients (ICC), typical error (TE) and typical error as a co-efficient of variation (%TE) were established. Normative strength data was determined using the most reliable measurement. An additional 30 elite athletes with a unilateral history of HSI within the last 12 months performed NHEs on the device to determine if eccentric weakness existed. The experimental device offers a reliable method to determine eccentric knee flexor strength and strength asymmetry

and was able to detect weakness in previously injured elite athletes similar to other methodologies.

6.3 INTRODUCTION

HSIs are the primary injury type in a number of sports, including Australian football,(7, 184) rugby union,(9) soccer(13, 15) and the sprinting events in athletics.(19) Compounding the issue further, HSIs are notorious for high rates of recurrence, which typically involve increased convalescence compared to the original insult.(9, 13) With respect to the mechanism of injury, a high proportion of HSIs occur during high speed running, with the terminal swing phase considered, by many, to be most injurious.(141) During terminal swing, the hamstrings are required to contract forcefully whilst lengthening to decelerate the flexing hip and extending knee.(37, 38) This combination of a high force contraction coupled with continually lengthening of the hamstring musculature likely contributes to the high incidence of HSIs during high speed running.(142) Not surprisingly, given the lengthening role of the hamstrings during the terminal swing phase, lower levels of eccentric knee flexor strength and between limb imbalances in eccentric knee flexor strength have been reported as a risk factor for future HSIs,(32, 33) indicating the importance of eccentric strength for HSI avoidance.(61, 143) Further to this, previously strained hamstrings display reduced levels of eccentric knee flexor strength compared to the uninjured contralateral limb,(43, 45) which may partially explain why previous HSI is the primary risk factor for future injury.(42) Recent observations of between limb eccentric strength asymmetries report the previously strained hamstring to be between 11-13% weaker than the uninjured limb.(45, 185)

Whilst the role of eccentric knee flexor function in the aetiology of HSI and reinjury is apparent, from a clinical perspective, one of the major hurdles that exist for

practitioners attempting to assess and monitor eccentric knee flexor strength in athletes is the lack of accessibility to an appropriate measurement device. Currently, the gold standard measure for the assessment of eccentric knee flexor strength is the laboratory based isokinetic dynamometer.(105) Whilst there are a number of benefits to isokinetic dynamometry, it is limited by the high cost of the device, it's inaccessibility and need for an experienced operator to ensure accurate and reliable data collection and analysis. As such, the isokinetic dynamometer is used largely for research purposes and occasionally by elite athletes or sporting teams as a means to screen individuals for HSI risk.(32) However, even in athletic environments the use of isokinetic dynamometry is, in our experience, met largely with resistance by sport and exercise science staff, who perceive an injury risk associated with the testing procedure. Further, in team sports, the amount of time required to complete a single athlete assessment (~30mins)(186) and the lab based nature of the equipment is prohibitive and logistically makes it difficult to assess the large number of athletes in a squad. As a result, despite increasing evidence as to the benefits of isokinetic strength assessment and monitoring for the prevention of HSI,(32, 33, 76) the application of this type of assessment in elite sports has been limited.

To enable a greater number of athletes to undergo eccentric knee flexor strength assessment, either on a one-off basis or as part of serial athlete monitoring, the major limitations of isokinetic dynamometry need to be overcome. Whilst hand held dynamometers have become popular in recent times they are very much dependent on operator skill to collected reliable and valid data.(186) Furthermore, particularly for the lower extremities, the operator must have sufficient strength to counteract the

force created by the athlete.(186) Here we propose a device which may overcome the limitations of both the isokinetic and hand held dynamometers. The experimental device utilises the commonly employed Nordic hamstring exercise (NHE) (Figure 6-1) to determine eccentric knee flexor force from each limb independently, under either bilateral or unilateral conditions. The device consists of padded braces used to secure the ankles with load cells located directly underneath and a base furnished with foam padding to provide cushion support for the knees (Figure 6-2). During the eccentric portion of the NHE the lower leg pulls up against the braces with the force transmitted axially and recorded by the load cells. This data can then be analysed by taking a single peak measurement or by averaging the peak from a series of contractions to determine eccentric knee flexor force and between limb force imbalances.

Whilst data exists pertaining to the reliability of eccentric knee flexor measures derived from the isokinetic(187-189) and handheld(186) dynamometers, no such information exists for the experimental device. In addition, data of normative values of the ratio of between limb eccentric knee flexor strength in uninjured individuals is required to determine if the device can detect eccentric strength asymmetries in previously injured hamstrings. The current study contains two investigations examining the experimental device. The purpose of the first investigation was 1) to determine the test-retest reliability of the variables measured via the experimental device using intraclass correlation coefficient (ICC), typical error (TE) and TE as a co-efficient of variation (%TE) to determine the best measure to use in future work; and 2) to determine normative levels of between limb eccentric strength asymmetries

in athletes with no history of HSI using the most reliable measure. The purpose of the second investigation was to compare strength asymmetry from the normative data set to retrospective data from elite athletes with a history of unilateral HSI in the last 12 months to determine if between limb asymmetries could be detected from the experimental device and whether the reported asymmetries are similar to what has been previously reported from isokinetic dynamometry.

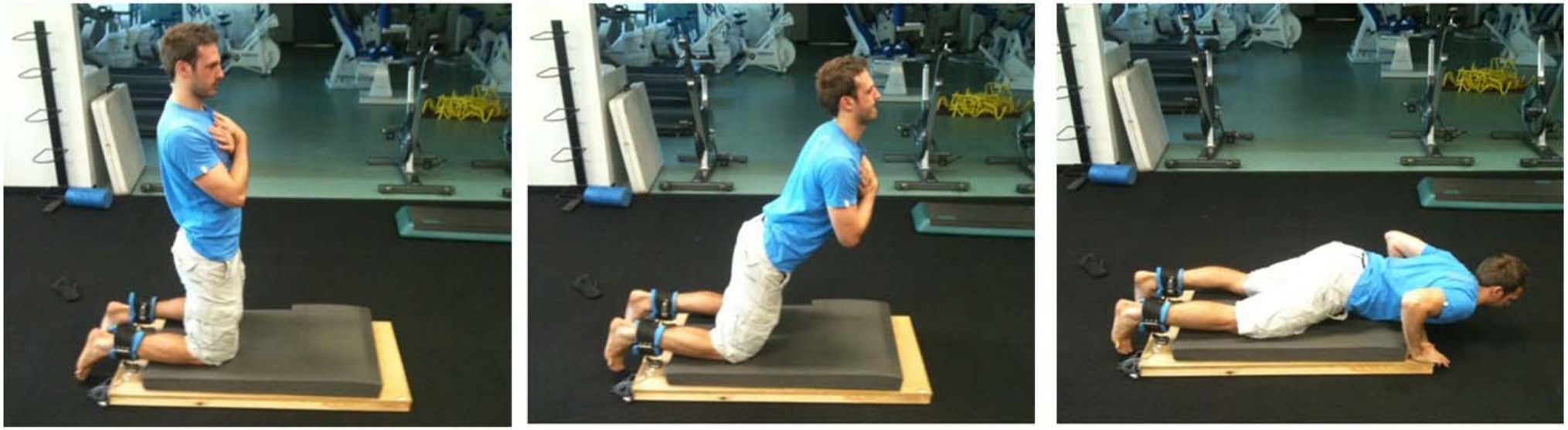
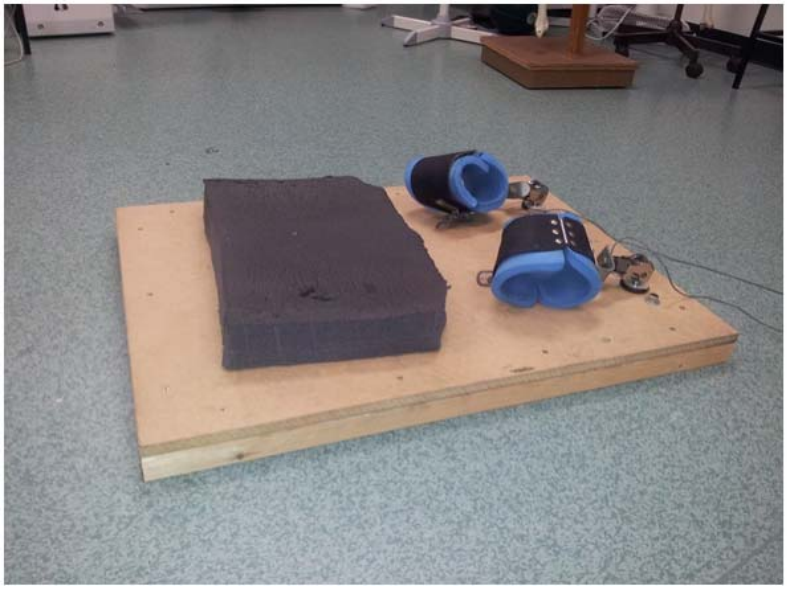


Figure 6-1. The performance of the Nordic hamstring exercise using the experimental device (progressing from left to right). The participant controls the speed of the fall by forceful eccentric contraction of the knee flexors. After the completion of the exercise the participant slowly returns themselves to the starting positions by pushing themselves back up with their hands (not shown). The ankles are secured independently in individual custom made braces.

A.



B.

Figure 6-2. The experimental device with A) individual ankle braces, padded cushion for knee support and wooden base; and B) close up view of the ankle brace, load cell organisation and pivot.

6.4 METHODS

6.4.1 PARTICIPANTS

For the first investigation 31 recreationally active males participated in the study, with most competing in Australian football, rugby (league, union or touch), soccer or sprinting. One participant was excluded from the study for continually changing their technique of the NHE between sessions, resulting in a total of thirty participants for analysis. For the second investigation 20 elite athletes, from Australian football, rugby union and track and field participated in the study. For both investigations all participants were free of any current injury to the lower limbs and were fully active in their chosen sport at the time of testing. All testing procedures were approved by the University Human Research Ethics Committee. Participants gave informed written consent prior to testing after having all procedures explained to them.

6.4.2 EXPERIMENTAL DESIGN

For the first investigation all participants reported to the laboratory on three separate occasions. The first session acted as a familiarisation session to prepare participants for all procedures to be performed in subsequent sessions and to correct any technique faults during the performance of the NHE. The following two sessions involved the determination of eccentric knee flexor strength via the experimental device. For the second investigation all participants were performing NHEs as part of their regular training routine, negating the need for a familiarisation session. As such these participants completed a single testing session of NHE with the experimental device to assess eccentric knee flexor strength.

6.4.3 EXPERIMENTAL DEVICE

For the first investigation participants were positioned in a kneeling position with padded foam underneath the knees for comfort (Figure 6-1). The ankles were secured by individual lockable braces which were affixed atop commercially available load cells (Transducer Techniques, CA, USA) (Figure 6-2). Following a warm up set of submaximal bilateral NHEs, participants were asked to perform two sets of three maximal NHEs bilaterally and unilaterally resulting in a total of four sets and 12 contractions per limb. Bilateral contractions were always performed before unilateral contractions, with the order of limbs tested unilaterally randomised between participants. The between set rest period was set at two minutes. The second investigation differed in that only bilateral NHE were performed because these were found to be more reliable than unilateral contractions (Table 6-1) and the load cells employed were custom made (Delphi Force Measurement, Gold Coast, Australia) and fitted with wireless data acquisition capabilities (Mantracourt, Devon, UK).

For both investigations participants were instructed to gradually lean forward at the slowest speed possible with the trunk held in a neutral position throughout (Figure 6-2) whilst the investigators gave verbal encouragement throughout the range of motion to ensure maximal effort. At the completion of the lowering phase the participants slowly returned themselves to the starting position by pushing themselves back up with their hands. Technique for all repetitions was monitored visually by the investigators and individual repetitions were rejected if they were not performed with correct technique.

6.4.4 INJURY HISTORIES

For all athletes recruited to the second investigation, details of their prior injury history for the preceding 12 months was ascertained from their club clinician. Details obtained included which limb was injured (dominant / non dominant limb), muscle injured (biceps femoris long head / biceps femoris short head / semimembranosus / semitendinosus), location of injury (proximal / distal, muscle belly / muscle-tendon junction), activity type performed at time of injury (i.e. running / kicking etc.) and grade of injury (I, II or III). Importantly, all diagnoses were confirmed by MRI performed 24-48 hours after the insult. Athletes who did not have an MRI performed, or who did not show a lesion on imaging, were excluded from the study.

6.4.5 DATA ANALYSIS

For the first investigation, force data for both limbs during the NHE was transferred to personal computer at 1000 Hz through a 16-bit PowerLab26T AD recording unit (ADInstruments, New South Wales, Australia). For both limbs (left / right) and conditions (bilateral / unilateral), the peak force for each contraction was determined and maximal force generating capacity was expressed as an average of the peak from six contractions (average peak force) and as the single highest peak of six contractions (peak force). Imbalance data, the ratio of between limb force from the experimental device, were presented as left limb: right limb ratio. Percentage differences, were calculated as recommended(187) using log transformed raw data followed by back transformation. For the second investigation, force data was transferred to personal computer at 100 Hz through a wireless USB base station receiver (Mantracourt, Devon, UK) with maximal force generating capacity

determined using the average peak method, which was shown, on the whole, to be the most reliable method to analyse the data.

6.4.6 STATISTICAL ANALYSIS

All statistical analyses were performed using JMP version 10.02 (SAS Institute, Inc). For the first investigation means and corresponding standard deviation for all force variables were reported for left and right limbs and for between limb force ratios. A spreadsheet by Hopkins(190) was used to calculate ICC, TE and %TE in order to determine the magnitude of variability from test 1 to 2.(191) We subjectively considered the data based on previously published quantitative guidelines where: an ICC $\geq$ 0.90 was regarded as high, between 0.80 and 0.89 as moderate and $\leq$ 0.79 as poor(192) and; a %TE of $\leq$ 10% was set as the level at which a measure was considered reliable.(193) Effect size (ES) was determined from test 1 and test 2 comparisons (test 1 minus test 2) to evaluate the magnitude of systematic bias. For reliability an ES (mean difference / pooled SD) of < 0.2 (the smallest worthwhile effect) was expected.

For the second investigation the previously injured elite athletes were compared to the normative data set using a restricted maximum likelihood method with the fixed factors being group (uninjured / injured) and limb (left / right or uninjured / injured depending on group) and the random factor being subject ID. Where significant effects were detected post hoc least squares difference (LSD) testing was used to identify which variables differed. Significance was set at $p < 0.05$ and effects size

(ES) was calculated using Cohen's *d*. Data are reported as mean difference and 95% confidence interval (95%CI).

6.5 RESULTS

6.5.1 INVESTIGATION 1

Descriptive statistics for all force variables for both test 1 and 2 are presented in Table 6.1 & 6.2. In addition, the magnitude of the differences from test 1 to test 2 is reported as an ES. One variable, bilateral peak force on the right leg, displayed a small detectable increase (ES = -0.20), whilst all other variables displayed no notable difference (Effect size = < 0.20). Table 6.1 & 6.2 also shows the test-retest reliability of all force variables from the experimental device. On the whole, using ICC as the measure of test-retest reliability, absolute force measurements taken during bilateral contractions (ICC ranged from 0.83 to 0.90) were more reliable than the unilateral condition (ICC ranged from 0.56 to 0.80). With respect to between limb strength (force) asymmetries, only the bilateral average peak force condition displayed acceptable reliability (ICC = 0.85, 95% CI = 0.71 to 0.93). Similarly, when using %TE to examine reliability, bilateral contractions (%TE ranged from 5.8 to 8.5) were more reproducible than unilateral contractions (%TE ranged from 7.9 to 11.0). Furthermore, between leg strength asymmetries were most highly reliable during the bilateral average peak force condition (%TE = 4.6, 95% CI = 3.7 to 5.9).

6.5.2 INVESTIGATION 2

The injury histories for all athletes can be found in Table 6.3. A main effect was detected for limb ($p < 0.0001$) with post hoc LSD testing determining that the previously injured limbs (319.7±100.3N) were 10% weaker compared to uninjured limbs (353.1±78.9N) (mean difference = 33.4N; 95%CI = 17.5 to 49.32N; $p < 0.0001$; ES = 0.62). Furthermore, an interactions effect was detected for group by

limb analysis (p=0.0378), with post hoc LSD testing determining that the previously injured limb was 15% weaker than the contralateral uninjured limb (mean difference = 56.3N; 95%CI = 25.7 to 74.9N; p = 0.0002; ES = 0.47), 15% weaker than the left (mean difference = 50.0N; 95%CI = 1.4 to 98.5N; p = 0.0437; ES = 0.60) and 18% weaker than the right limb (mean difference = 66.5N; 95%CI = 18.0 to 115.1N; p = 0.0080; ES = 0.79) from the uninjured normative group (Table 6.4). No differences existed between the uninjured limb from the injured group and the left (p = 0.9891) and right limb (p = 0.5064) from the normative uninjured group (Table 6.4). No main effect was detected for group (p=0.1554).

Table 6-1. Descriptive statistics and test-retest reliability data for variables derived from the experimental device using peak force measurements (n=30).

	Test 1 Mean±SD (N)	Test 2 Mean±SD (N)	ES	ICC (95% CI)	TE (95% CI) (N)	%TE (95% CI)
Bilateral peak force						
Left Leg	366.4±67.7	374.1±60.5	-0.10	0.83 (0.67 to 0.91)	27.5 (21.9 to 36.9)	8.5 (6.7 to 11.6)
Right Leg	378.4±68.4	391.6±67.0	-0.20	0.90 (0.81 to 0.95)	21.7 (17.3 to 29.2)	5.8 (4.6 to 7.9)
Imbalance (left: right)*	0.97±0.11	0.96±0.12	0.19	0.76 (0.55 to 0.88)	0.06 (0.05 to 0.08)	6.0 (4.8 to 8.2)
Unilateral peak force						
Left Leg	351.3±55.5	356.8±65.6	-0.07	0.73 (0.51 to 0.86)	32.3 (25.7 to 43.5)	10.2 (8.1 to 14.0)
Right Leg	380.9±60.4	370.4±54.7	0.09	0.56 (0.26 to 0.76)	38.8 (30.9 to 52.1)	11.0 (8.7 to 15.1)
Imbalance (left: right)*	0.92±0.13	0.96±0.13	-0.16	0.40 (0.05 to 0.66)	0.09 (0.07 to 0.11)	10.1 (8.0 to 13.9)

SD, standard deviation; ES, Cohen's *d* effect size; 95% CI, 95% confidence interval; N, Newtons; ICC, intraclass correlation coefficient; TE, typical error; %TE, typical error as a coefficient of variation. *Imbalance data expressed as a ratio and not in Newtons. Peak force is the highest maximal force recorded from six contractions. Average peak force is the mean of maximal force recorded from six contractions.

Table 6-2. Descriptive statistics and test-retest reliability data for variables derived from the experimental device using average peak force measurements (n=30).

	Test 1 Mean±SD (N)	Test 2 Mean±SD (N)	ES	ICC (95% CI)	TE (95% CI) (N)	%TE (95% CI)
Bilateral average peak force						
Left Leg	336.3±63.8	344.7±61.1	-0.09	0.85 (0.71 to 0.93)	24.7 (19.7 to 33.2)	8.4 (6.6 to 11.5)
Right Leg	349.4±64.8	361.2±65.1	-0.16	0.89 (0.78 to 0.95)	22.1 (17.6 to 29.7)	6.5 (5.1 to 8.8)
Imbalance (left: right)*	0.96±0.11	0.95±0.13	0.13	0.85 (0.71 to 0.93)	0.04 (0.04 to 0.06)	4.6 (3.7 to 5.9)
Unilateral average peak force						
Left Leg	321.4±54.0	323.6±64.2	0.01	0.79 (0.61 to 0.90)	27.6 (22.0 to 37.2)	9.5 (7.5 to 13.0)
Right Leg	341.8±50.9	335.8±54.7	0.11	0.80 (0.63 to 0.90)	24.1 (19.2 to 32.5)	7.9 (6.2 to 10.7)
Imbalance (left: right)*	0.94±0.12	0.96±0.14	-0.08	0.55 (0.24 to 0.76)	0.08 (0.06 to 0.10)	8.7 (6.9 to 11.9)

SD, standard deviation; ES, Cohen's *d* effect size; 95% CI, 95% confidence interval; N, Newtons; ICC, intraclass correlation coefficient; TE, typical error; %TE, typical error as a co-efficient of variation. *Imbalance data expressed as a ratio and not in Newtons. Peak force is the highest maximal force recorded from six contractions. Average peak force is the mean of maximal force recorded from six contractions.

Table 6-3. Details of injury histories for elite athletes from Australian football, rugby union and track and field with a unilateral hamstring strain injury within the prior 12 months.

Subject	Limb	Muscle	Location	Grade	Injurious activity	Time since injury (months)
1	Dominant	SM	Proximal MTJ	1	Running	2.5
2	Non-dominant	BFLH	Proximal MTJ	1	Running	3
3	Non-dominant	BFLH	Mid muscle belly	2	Running	3
4	Non-dominant	SM	Distal MTJ	1	Running	3.5
5	Non-dominant	BFLH	Mid muscle belly	1	Not defined	3.5
6	Non-dominant	SM, ST	Proximal muscle belly	1	Running	2
7	Non-dominant	BFLH	Proximal MTJ	1	Running	8.5
8	Non-dominant	BFLH	Proximal muscle belly	1	Running	4
9	Non-dominant	BFLH	Proximal MTJ	1	Running	5
10	Non-dominant	BFLH	Proximal muscle belly	2	Jumping	7.5
11	Dominant	BFLH	Mid muscle belly and distal	2	Running	6.5
12	Dominant	BFLH	MTJ	2	Running/bent	7
13	Dominant	BFLH	Proximal MTJ	1	forward	8
14	Dominant	BFLH	Proximal MTJ	1	Running	10
15	Non-dominant	SM	Distal MTJ	1	Running	11
16	Non-dominant	BFLH	Proximal MTJ	1	Stretching	11
17	Dominant	BFLH	Distal muscle belly	2	Running	8
18	Dominant	SM	Proximal MTJ	2	Running	3
19	Non-dominant	BFLH	Proximal MTJ	1	Bent forward/pushed	2.5

BFLH, biceps femoris long head; MTJ, muscle-tendon junction, SM, semimembranosus; ST, semitendinosus.

Table 6-4. Comparison of eccentric knee flexor strength derived from the experimental device in athletes with and without a history of unilateral hamstring strain injury within the prior 12 months.

	Control (normative) group		Injured group	
	Left limb	Right limb	Uninjured limb	Injured limb
Eccentric knee flexor strength (N)	344.7±61.1	361.2±65.1	351.0±78.9	294.7.2±100.3[a]

[a] significantly weaker than all other limbs. Significance set at $p < 0.05$.

6.6 DISCUSSION

Although the incidence and recurrence rates of HSIs in a number of sports has not declined in many years,(142) there has been a proliferation of scientific literature focused on the prediction, prevention and rehabilitation of this injury. Of most prominence in recent times is an appreciation for the importance of greater eccentric knee flexor strength to reduce the risk of future hamstring strain injury,(32, 33, 61, 143) particularly given recent reports which indicate that eccentric knee flexor strength is compromised in previously injured athletes despite return to competition.(45, 185, 194) However at a practical level, the measurement of eccentric knee flexor strength in athletic populations is limited by current (isokinetic or handheld) dynamometry methodologies. These limitations include, but are not limited to, high device costs, lack of accessibility, long assessment times and the skill and strength of the clinician.(186) The experimental device used in the current study offers a field based assessment tool which allows time-efficient measurement of eccentric knee flexor force that is largely independent of an operators skill. Importantly, the exercise used to collect this information, the NHE, is one commonly employed in elite sport.(32, 33, 61, 143) The present study aimed firstly to examine the test-retest reliability of the experimental testing device and determine normative values for eccentric knee flexor strength in uninjured athletes. The second aim of the study was to examine if elite athletes with a previous history of HSI within the prior 12 months had any strength deficits in their previously injured limb that could be detected by the experimental device.

The major findings from the current study are 1) the experimental device displayed high to moderate test-retest reliability when the NHE was performed bilaterally, but poor reliability during unilateral contractions; 2) recreational athletes with no history of lower limb injury displayed no significant eccentric knee flexor strength differences between the left and right limbs and; 3) elite Australian football, rugby union and track and field athletes with a unilateral history of HSI within the last 12 months displayed significant eccentric knee flexor weakness in their injured limb compared to their uninjured limb and to uninjured recreational athletes.

From the data presented, the experimental device displays high to moderate levels of test-retest reliability when measuring peak or average peak knee flexor force during a bilateral NHE and approaches a moderate level of reliability for average peak force during unilateral contractions. For the measurement of between limb strength differences only when the NHE was completed bilaterally, and peak force was average across multiple contractions, did the measure display moderate reliability (ICC = 0.85). It should be noted that the ES for the right limb bilateral peak force was equal to -0.20, which may be indicative of a learning effect from test 1 to test 2. Such a learning effect is not present in any other of the assessed variables so it remains unknown why only the right limb during a bilateral NHE assessed using a single peak force measure displayed such a trend. Both the isokinetic (ICC range from 0.83 to 0.97)(187-189) and handheld dynamometers (ICC = 0.90)(186) have reported similar or slightly higher levels of reliability for assessing eccentric knee

flexor strength compared to the most reliable measure reported from the experimental device (bilateral average peak force, ICC = 0.85 to 0.89). To our knowledge only one other previous study has examined the reliability of between limb eccentric knee flexor strength imbalances and reported low test-retest reliability (ICC = 0.69)(189), which is well below the most reliable measure reported here for the experimental device (bilateral average peak force, ICC = 0.85). By means of a secondary measure, a %TE ≤ 10.0% was considered a reliable measure, as per previous work.(193) Use of %TE confirms that bilateral contractions (TE% ranged from 5.8 to 8.5) are more reliable than unilateral contractions (TE% ranged from 7.9 to 11) and that the bilateral average peak force condition was the most reliable method for obtaining between limb imbalance data (TE% = 4.6, 95% CI = 3.7 to 5.9). These findings indicate that the single most reliable method to acquire eccentric knee flexor force and between limb force ratios from the experimental device is via a bilateral NHE with peak force averaged across multiple contractions.

To the knowledge of the authors, the second investigation is one of the largest retrospective hamstring strain injury studies utilising elite athletes. A further strength to this study compared to previous retrospective work is that all previously injured athletes had the diagnosis of a HSI confirmed by MRI, which eliminates the inclusion of athletes with referred pain posterior thigh injury.(20) The finding that a previously strained hamstring still displayed eccentric knee flexor weakness compared to the contralateral uninjured limb despite ‘successful’ rehabilitation confirms recent work which has utilised isokinetic dynamometry to assess strength in sub-elite populations.(45, 185) Indeed, the percentage difference in eccentric strength

between limbs reported here in the injured cohort (15%) is in line with previous data using isokinetic dynamometry examining athletes with a prior history of unilateral hamstring strain injury (11-13%).(45, 185) Future research should determine if the eccentric knee flexor force measures derived from the experimental device are predictive of athletes at an elevated risk of hamstring strain injury. Prospective studies determining whether preseason measures of eccentric knee flexor strength and between limb strength asymmetry using the experimental device can predict athletes at an elevated risk of future injury should be considered. The ability of the device to be used as a serial measure of eccentric hamstring strength across a season should also be investigated.(5)

It may be argued that it would have been appropriate to normalise the force measurements derived from the experimental device to the stature and weight of the participant, however we don't believe this to be critical. Due to the nature of the NHE all athletes reach a critical point in the range of motion, where the ever increasing external load from gravity acting on the upper body, exceeds the maximal eccentric hamstring strength of the athlete. Whilst the position in the range of motion where this critical point occurs will be influenced by individual anthropometric characteristics and strength level, by reaching this critical point it will require maximal force generation of the knee flexors. As such the NHE is an excellent vehicle for assessing absolute eccentric knee flexor strength without the need for normalising to height or weight. The major limitation of the current study relates to the retrospective nature of the second investigation. With this study design it is not possible to determine whether the eccentric weakness seen in the previously injured

limb was the cause or the result of injury. It should be noted, however, that prospective studies have determined that a between limb eccentric strength difference of approximately 4.5% is associated with future hamstring strain injury,(33) whilst post-injury eccentric weakness is reported to be between 11-13%,(45, 185) suggesting hamstring injury enhances eccentric knee flexor weakness.(185) Therefore, it might reasonably be expected that the deficits in eccentric knee flexor strength are at least partly due to previous injury.

In conclusion, a novel field testing device using the NHE as a vehicle to measure eccentric knee flexor strength and between limb strength asymmetry has demonstrated high to moderate levels of test-retest reliability during bilateral contractions. The device also displayed an ability to detect eccentric weakness in a previously injured limb of elite athletes to a similar extent as previous methodologies. This portable device offers an alternative to current dynamometry based techniques for the assessment of eccentric knee flexor strength. Given the importance of eccentric knee flexor strength in the aetiology of hamstring strain injuries, greater accessibility of a reliable assessment tool to measure this strength quality might be expected to assist sports science and sports medicine practitioners in determining the level of hamstring strain injury risk and implementing appropriate preventative strategies for individual athletes.(32) However, further work is required to determine the predictive ability of the experimental device and how it might be best utilised to assist with a hamstring strain injury prophylactic program.

7 GENERAL DISCUSSION AND CONCLUSIONS

The primary purpose of the thesis was to examine the impact of intermittent running and previous hamstring strain injury (HSI) on the neuromuscular function of the knee flexor muscle group. The focus on these two conditions is because: 1) the incidence of HSIs increase during the later portions of halves in intermittent running-based sports such as rugby union(9) and soccer(15) which implies that fatigue is a potential causative factor in HSI; and 2) previous HSI is the primary risk factor for future HSI(42) suggesting that neuromuscular maladaptations arising from a prior insult may leave the injured tissue vulnerable to future damage.(142) Furthermore, given the largely eccentric-specific nature of the declines in neuromuscular function following intermittent running and previous HSI and the lack of accessibility to a valid and reliable methodology for the assessment of eccentric knee flexor strength, a secondary focus of this thesis was to developed a suitable, novel device to measure this strength quality.

The primary novel findings arising from this thesis are:

Study 1

- The knee flexor angle of peak torque does not influence the magnitude of the decline in eccentric knee flexor strength following 45 minutes of intermittent running;

- the decline in eccentric knee flexor strength following intermittent running is highly variable between individuals.

Study 2

Compared to the contralateral uninjured limb, a limb which had previously suffered a HSI to biceps femoris long head (BF_L) displayed:

- lower levels of knee flexor strength across fast and slow concentric and eccentric contractions;
- a lower level of electromyography (EMG) activity specifically in BF_L during slow and fast eccentric contractions, whilst concentric contractions and the medial hamstrings (MH) remained unaffected;
- no difference in the median power frequency of the EMG signal during concentric or eccentric contractions in the BF or MH.

Study 3

Compared to the contralateral uninjured limb, a limb which had previously suffered a HSI to BF_L displayed:

- lesser rate of torque development (RTD) and impulse (IMP) 50 and 100 ms after the onset of an anticipated slow eccentric contractions;
- lesser BF_L EMG activity at 100 ms after the onset of EMG activity in anticipation of fast and slow eccentric contractions
- no difference in the EMG activity of the MH.

Study 4

A novel field testing device, designed to assess eccentric knee flexor strength by utilising the NHE displayed:

- mostly acceptable test-retest reliability during bilateral, but not unilateral, contractions;
- the ability to detect eccentric weakness in a previously injured limb, to a similar magnitude as previously validated methodologies.

Emanating from the initial studies in this thesis is further confirmation as to the importance of eccentric strength of the knee flexor muscle group in the aetiology of HSI, with both fatigue and previous HSI shown to impact, mostly, upon eccentric strength and EMG activity. Both of these responses present possible explanations as to why fatigue and previous HSI may be implicated in the injury process. The key question that remains following these findings is: what are the mechanisms responsible for these deficits in eccentric knee flexor neuromuscular function? A question which in itself requires further thorough investigation. With respect to eccentric knee flexor specific weakness consequent to intermittent running, unpublished observations from our laboratory have shown that declines in EMG activity of the BF_L can partially explain the decline seen in isokinetic eccentric knee flexor strength at $-180^{o}.s^{-1}$. This data suggests that central factors may mediate the eccentric specific declines in knee flexor function following intermittent running, which is somewhat similar to what has been reported in this thesis for the previously injured hamstring. In both cases, further investigation into where in the motor

pathway the deficits in neural function are occurring, is of critical importance if effective interventions are to be developed. This would require the use of techniques such as twitch interpolation and transcranial magnetic stimulation to localise spinal and supraspinal deficits in neural function, however applying these methodologies to the hamstring muscular would require further development of the procedures given the dearth of literature reporting the use of these approaches on the hamstrings/knee flexors.

The invention of an accessible field testing device to measure eccentric knee flexor strength has the potential to impact significantly on the field of HSI prevention and rehabilitation. Greater accessibility than the current gold standard of isokinetic dynamometry will allow a greater number of athletes to undergo assessment of eccentric knee flexor strength and between limb strength asymmetries, which would be expected to assist with more targeted and individualised interventions. Furthermore, the experimental device could have applications for informing the rehabilitation process with more objective data than is currently available, by using the device to ensure any between limb strength asymmetries are normalised before rehabilitation is considered to be complete and athletes are cleared to return to play. To further explore the potential applications of the experimental device, retrospective studies comparing between limb eccentric knee flexor strength asymmetry in athletes with a previous unilateral HSI would be warranted. Further, prospective studies to determine if the measures derived from the experimental device are able to predict which athletes go on to sustain a future HSI would inform as to what level of between limb strength asymmetry, if any, should be considered ‘at risk’. Also given

the findings from Study 3 in the current thesis, if the experimental device is able to measure rate of force development, this may give the device further scope to assist with the prevention and rehabilitation of HSIs.

The information emanating from this thesis gives a clear direction for future work. In particular, deficits in neural function in previously injured hamstrings were identified using surface EMG (Chapter 4 & 5), which measures grossly the neural drive delivered to the muscles of interest. Other available methodologies, such as twitch interpolation and transcranial magnetic stimulation, can be used to determine whether these neural deficits are emanating from the spinal or supraspinal centres. These approaches should be investigated further in the future. Furthermore, the neuromuscular deficits in hamstring function following HSI may have the potential to impact upon the risk of other injury types as well. Given the important role the hamstring muscle group plays in dynamic stability of the knee, lingering neuromuscular deficits in hamstring function may impose an increased risk of sustaining a traumatic knee joint injury such as an anterior cruciate ligament rupture. More work needs to be done to determine if a prior HSI impacts on knee joint stability during potentially injurious activity types and movements. Finally, the experimental device presented in Chapter 6 (see Figure 6-1 & 6-2) is currently being utilised in a large (n=250) prospective study in Australian football, to determine the predictive ability of the eccentric strength measurements derived from the device.

In conclusion, the current thesis has determined a raft of deficits in fatigued and previously injured hamstrings that are mostly confined to eccentric contraction

modes. These findings have important implications given the agreed upon role of eccentric contraction in the aetiology of HSIs within the literature. Extending from these findings, a novel experimental device has been proposed as an alternative to current methods used to assess eccentric knee flexor strength that is both easily accessible and reliable. Further work in these areas is crucial for the ongoing efforts to minimise the incidence and recurrence of HSIs.

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APPENDICIES

APPENDIX 1: Published paper that forms the basis of Chapter Two

APPENDIX 2: PUBLISHED PAPER THAT FORMS THE BASIS FOR CHAPTER FOUR

APPENDIX 3: PUBLISHED PAPER THAT FORMS THE BASIS OF CHAPTER FIVE